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INJURY IN SPORT

Injury in Sport

The Physiology, Prevention and Treatment of Injuries associated with Sport

EDITED BY

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FOREWORD BY

SIR ADOLPHE ABRAHAMS

O.B.E., M.D., F.R.C.P.

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FOREWORD

IT IS an anomaly that this country, rightly regarded by the rest of the world as the pioneer of sport, should be behind in attention to the injuries and other afflictions inevitably encountered by athletes, in contrast to the practice throughout the Continent and the United States where every club or organisation has its 'sports doctor'. In many instances his is a whole time appointment appropriately subsidised, and there are examples of such elaborateness of administration as a Professor of Physical Culture at the head of a team of assistants, physiotherapists, trainers, coaches and everything devoted to athletic well being.

Not that this country lacks the personnel. There are a number of medical men with the athletic interest and experience well known for their services, of course in an honorary capacity but necessarily handicapped by the lack of financial aid and—it must be admitted—the lack of encouragement on a national scale and of what may be regarded as official recognition.

Among our authorities, Bill Tucker is outstanding. He adds to athletic prowess and distinction (he captained Cambridge at rugger and was an English International) an unparalleled enthusiasm and inexhaustible enterprise in the investigation and treatment of athletic injuries. J. R. Armstrong, the author of two famous orthopaedic monographs, *Lumbar Disc Lesions* and *Bone Grafting*, and he have edited this remarkable symposium, remarkable in that they have enlisted the co-operation of physicians and surgeons interested and experienced in every branch of sport from Association Football to Winter Sports who have dealt individually with the injuries encountered. They have gone further; they have secured the services of some of the greatest performers who have contributed their views as practical exponents. Among these may be recognised: Miss Pat Smythe, Dan Maskell, Jim Peters, Denis Compton, to name only a few of world repute.

The subject is introduced, as is proper and logical, with details of the anatomy, physiology and psychology of the normal. Mr Tucker himself deals with posture and the mechanics of movement, subjects in which he has always exhibited special interest; after which the separate categories of sport are left in the hands of doctor and athlete as individual pairs.

A final section describes the diagnosis and treatment of every form of injury. Mr Tucker has been responsible for soft tissue lesions throughout the body, Mr Armstrong for fractures and spinal injuries. In all an ambitious, highly successful undertaking which it is no exaggeration to say is of national importance comprising something which no other country could have pro-

duced and appropriate to the princes of sport who may well feel proud in maintaining a claim to lead the world.

ADOLPHE ABRAHAMS

CONTENTS

FOREWORD: Sir Adolphe Abrahams, O.B.E., M.D., F.R.C.P. *Page* v

PREFACE: J. R. Armstrong, M.D., M.CH., F.R.C.S. xi
W. E. Tucker, C.V.O., M.B.E., T.D., F.R.C.S.

PART I

1. Body Structure in Relation to Limb Movements 1
K. A. Provins, PH.D., M.A. (Oxon)

2. Physiological Factors Limiting Maximum Performance 33
J.E. Cotes, M.A., B.M., B.Ch., M.R.C.P.

3. Posture and the Mechanics of Movement 62
W. E. Tucker, C.V.O., M.B.E., T.D., F.R.C.S.

4. Fitness and Training 82
W. E. Tucker, C.V.O., M.B.E., T.D., F.R.C.S.

5. The Significance of Somatotype in Sport and Athletics 94
Ian J. MacQueen, M.B., B.S., F.R.C.S.E., F.R.C.S.

6. Physiological and Psychological Approach to Athletics 105
D. J. Cussen, M.D.

7. Women in Sport 113
A. H. Charles, E.R.D., T.D., M.A., F.R.C.S., F.R.C.O.G.

8. Nutrition and the Athlete 123
Raymond Dixon Firth, M.R.C.S., L.R.C.P.

PART II

1. Mechanism of Production of Injury 133
J. R. Armstrong, M.D., M.Ch., F.R.C.S.

2. Association Football 137
Kevin O'Flanagan, M.B., B.Ch., Arsenal and England
Jimmy Hill, Manager, Coventry City

3. Injuries to Athletes 142
Sir Adolphe Abrahams, O.B.E., M.D., F.R.C.P.
Jim Peters, British Marathon Record Holder

4. Baseball 148
The late George E. Bennett, M.D.

5. Baseball Injuries 151
Edward E. Kimbrough, M.D.

6. Basketball 155
Emmett M. Lunceford, Jun., M.D.

7. Boxing Injuries 159
J. L. Blonstein, M.R.C.S., L.R.C.P., D.I.H.
Henry Cooper, British and Empire Heavyweight Champion

8. Injuries at Cricket 168
N. Vere-Hodge, F.R.C.S.
Denis Compton, Middlesex and England

9. The Fencer at Risk 173
R. Parfitt, M.R.C.S., L.R.C.P., L.D.S., D.M.R.T., F.F.R.

10. Game Shooting and Injury 191
J. R. Armstrong, M.D., M.Ch., F.R.C.S.
Joseph Nickerson

11. Golf 200
I. H. M. Curwen, M.B., Ch.B., D.Phys. Med.
Tom Haliburton, British Ryder Cup Team

12. Injuries in Hockey 206
Norman Borrett, Captain of the English Hockey Team

13. Riding Accidents 210
R. P. Goulden, M.B., Ch.B., D.Phys.Med.
Pat Smythe, International Show Jumping Champion

14. Tennis Elbow 216
M. Slapak, M.A., M.B., B.Ch.
Dan Maskell, O.B.E., British Lawn Tennis Association Training Manager.

15. Motor Racing Accidents 224
W. Carton Winterbottom, L.M.S.S.A.

16. Injuries in Rowing 226
Raymond Owen, M.A., M.B., B.Ch., D.R.C.O.G., M.M.S.A.

17. Rugby Football 230
J. W. Kyle, M.B., B.Ch.
Peter Wright, England

18. Swimming 236
P. Hume Kendall, M.R.C.P., D.Phys.Med.

19. Water Ski-ing 242
Austin T. Moore, M.D.

20. Winter Sports 250
John A. Williams, Ch.M., F.R.C.S.

21. Ski-ing 259
Colonel B. J. Murphy, Ski Club of Great Britain

PART III

SECTION I. *Soft Tissue Injuries*, W. E. Tucker, C.V.O., M.B.E., T.D., F.R.C.S.

1. The Diagnosis of Soft Tissue and Joint Injuries 263

2. Pathology 274

3. Treatment 288

4. The Classification of Injuries and their Treatment 318
Injuries to Arteries
Sol Cohen, M.A., F.R.C.S. 339

5. Upper Limbs 359

6. Head, Neck and Trunk 405

7. Lower Limbs 414

8. A Critical Survey of Methods Employed in the Treatment of Athletic Injuries: The Difficult Case to Cure 445

9. Complications following Athletic Injuries 455

SECTION II. *Fractures in Athletes*, J. R. Armstrong, M.D., M.Ch., F.R.C.S.

1. The Production and Healing of Fractures 468

2. The Principles of Treatment of Fractures 493

3. Fractures of the Different Body Regions 512

4. Fractures of the Different Body Regions (continued) 524

5. 'Stress' or 'Fatigue' Fractures 533

SECTION III. *Spinal Injuries in Athletes*, J. R. Armstrong, M.D., M.Ch., F.R.C.S.

1. Structure of the Spinal Column 550

2. The Spine in Movement and Posture 561

3. Traumatic Lesions of the Spine 575

4. Traumatic Lesions of the Spine (continued) 583

5. Intervertebral Disc Lesions 593

INDEX 606

PREFACE

UNLESS there is a nuclear catastrophe, it seems certain that during the next fifty years one of the major problems facing mankind will be the employment of leisure. Increasing mechanisation and industrial efficiency must ultimately leave everyone with time on their hands.

In these circumstances various things might happen. It might be that the vast majority of people would demand to be entertained and this demand would be catered for by a small group of professionals which would, of course, include professional athletes of all kinds. The undesirability of such a state of affairs is obvious and, happily, there are already clear indications that most people would wish to employ their spare time by participating actively in some sport or other pastime. Huge problems will arise if the majority of people wish to participate rather than watch, for example, the difficulties and expense of building a stadium to house 100,000 spectators are nothing compared with the problems of providing facilities for 100,000 people to take active part in various games and sports. Nevertheless, with the coming of ample leisure these problems must be faced and overcome.

One of the points which is certain to demand attention is the risk of physical injury associated with all sorts of recreations. There is, of course, no sport which is entirely without risk and, indeed, it has been claimed that an element of danger might be an attraction. This is highly doubtful, especially from the participants point of view, and certainly no professional athlete will willingly incur avoidable risk. It is also doubtful whether the element of danger is desirable from the spectator's point of view—humanity has progressed a little since the days of the gladiators and bear baiting.

In the future, therefore, it is going to be the concern of an increasing number of people to render all forms of sports safer. This book is an examination of the injuries which may occur in sports of all kinds. It is true that the large part of it is occupied with the nature and management of these injuries but this must lead to an understanding of how injuries occur and how they may be prevented. If it succeeds in rendering sport a little safer and in decreasing the amount of permanent or temporary disability following inevitable accidents it will have served its purpose.

No book in one volume can contain everything that can be said on this subject. There are bound to be certain omissions due to lack of space and many injuries are not discussed in detail. In such instances the reader can

amplify his knowledge from standard textbooks and many such references are given.

We are grateful to our many friends and acquaintances who have allowed us to use material from their work, both as quotations and in the form of figures and diagrams. We also tender our thanks to the following publishers for permitting us to reproduce diagrams and illustrations from medical works published by them: Ernest Benn Ltd (*Hand Atlas of Human Anatomy*, 1900): Blackwell Scientific Publications Ltd (D. Sinclair: *An Introduction to Functional Anatomy*): Dr P. A. Merton and J. & A. Churchill Ltd ('Speculations on the servo-control of movement' in CIBA Foundation Symposium on *The Spinal Chord*; 1953): Her Majesty's Stationery Office (Medical Research Council Memorandum No. 20—*The Physique of Young Adult Males*): E. & S. Livingstone Ltd (G. A. G. Mitchell and E. L. Patterson; *Basic Anatomy*, 1954): Longmans, Green & Co. Ltd (E. G. Walsh; *Physiology of the Nervous System*, 1957): Oxford University Press (Samson Wright; *Applied Physiology*, 1955): to Dr Katharine Wells of Mary Washington College of the University of Virginia for permission to use material from her *Kinesiology*, published by W. B. Saunders Company, Philadelphia, USA., and to Sir Wilfred le Gros Clark for permission to reproduce two figures from the fourth edition of his book, *The Tissues of the Body*, published by The Clarendon Press, Oxford.

We wish to acknowledge the help that we have had from many people in the preparation of this book. Group Captain Dhenim, J. Mahoney, F.R.C.S., Dr A. B. Corrigan, Dr R. P. Goulden and Dr W. Hargreave-Wilson have been of the greatest assistance. We would like to thank our artists, Miss Fairfax-Whiteside and Mr R. N. Lane. Our contributors and our publishers have been unfailingly patient with the many delays caused by other demands on our time. Finally, without the work of our secretaries, Miss A. M. Carding and Miss P. E. Timson this book would probably never have been completed.

J. R. ARMSTRONG
W. E. TUCKER

London : January 1964

PART I

1

BODY STRUCTURE IN RELATION TO LIMB MOVEMENTS

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INTRODUCTION

In attempting to understand the way in which skilled movements are made, it is first of all essential to know something of the various structures involved; for the design and composition of the moving parts determine to a very large extent the type of movement which it is possible to make. The strength, range and accuracy of movements of different body members in any given direction are dependent on a number of factors which are discussed in the following sections. The variation in strength, range and accuracy of movements of the same body members in different people is also briefly considered.

Structural features do, of course, vary from person to person and this is simply an example of biological variation. In the same way that people vary in height and weight (i.e. general body size), so their component parts may vary or the internal organisation of the parts relative to one another. Fig. 1 shows the frequency of occurrence of each measurement of one particular dimension of body size in a sample of the normal British population. It should be noted that there is a continuous variation in size from very small to very large. There are a few people at the extremes but most are included within definable limits. In a normal distribution, 68 per cent of the observations lie within a distance equal to the standard deviation* on each side of the mean, while plus or minus twice the standard deviation includes 95 per cent and plus or minus three times the standard deviation includes 99·7 per cent of the observations. Human anatomy is the study of those structural characteristics which are common to man, taking into account normal variation.

The structures mainly concerned in making movements are bones, muscles and nerves, which together form the Locomotor System. All movements are

* The standard deviation is a statistical measure of scatter and may be expressed as 'the square root of the mean of the squares of the deviations of the observations from their arithmetic mean': (see Chambers, 1946, p. 17).

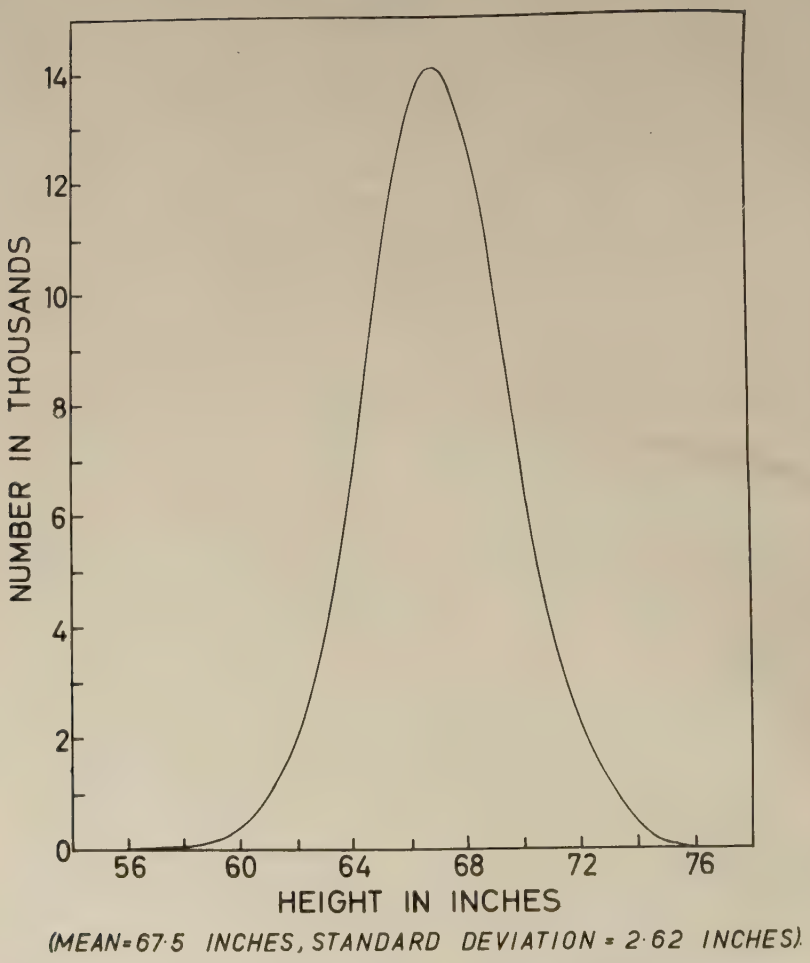

FIG. 1. Height distribution of a sample of 91,163 males aged twenty years (data taken from W. J. Martin; 'The physique of young adult males'; *Medical Research Council Memorandum No.* 20; London, H.M.S.O. 1949).

initiated by nerve impulses which cause contraction of the muscles acting about the joints. Consequently the structural limitations of these components considered separately and their organisation in any particular situation will primarily determine the limitations of the ensuing joint movement. The precise nature of these limitations is made clear in the following sections.

Bones

Bones form the skeletal framework of the body. They provide for the other living tissues a system of support which resists gravity and enables an upright posture to be maintained. In other words, bones give the human body rigidity which not only allows it to resist external forces, but also to exert forces against objects in the environment.

Some of the lower animals, such as worms, have no bony skeleton at all; their mode of progression is slow and they are very much at the mercy of their environment. Others, such as insects, and crustaceans like the crab or lobster,

FIG. 2. Longitudinal section of bone showing A, outer compact bone enclosing B and C, spongy or cancellous substance with delicate interlacing bony plates (lamellae).

have an exoskeleton; that is, their supporting framework is on the outside and their muscles and other tissues are completely enclosed. While this arrangement makes for much improved methods of locomotion, it is very clumsy, and severely restricts the range of movements possible at any one joint. Furthermore, the size of the muscular tissue of the limbs is limited by the internal dimensions of the skeleton. In vertebrates, however, the muscles are attached to the outside of the bony framework of the limbs, an arrangement which, in man, with his upright posture, allows considerable freedom of movement of the hands and arms. Other bones such as the skull and ribs which do enclose certain organs of the body primarily serve a protective function.

The internal architecture of a limb-bone sectioned longitudinally is shown in Fig. 2. There is an outer shell of a relatively dense nature, called compact bone, which gradually gives way to bone of a much more open texture (spongy bone) at the centre. The shaft of the bone is thus a hollow tube, combining strength and rigidity with lightness. The arrangement of the bony plates forming the open meshwork of spongy bone is closely related to the mechanical forces which the bone is called upon to withstand during growth. This can be seen in Fig. 3 which is a longitudinal section through the upper part of the femur or thigh bone. The lattice-work of bony plates reflects the lines of stress and strain to which the bone has been subjected; and their arrangement has frequently been likened to the pattern of iron girders in a crane or bridge. As a result of injury or deformity, the internal architecture of a bone may be considerably modified. Much information now exists on the breaking strength of bones. The interested reader is referred to a recent monograph on the subject by F. Gaynor Evans (1957).

Bone is mainly composed of inorganic salts which make it the most durable tissue in the body apart from the enamel and dentine of the teeth. These salts (chiefly calcium phosphate) are deposited round a tough network of inelastic tissue (collagen fibres) and form a reservoir for calcium in the body. In some bones, the interstices of the spongy bone contain the red marrow responsible for manufacturing the red cells which circulate in the blood and act as oxygen carriers. Covering the bone shaft everywhere, except at the joint surfaces, is a tough fibrous membrane called the periosteum which provides the means of attachment of muscles, tendons and ligaments to the underlying bone.

Joints

The junction of one bone with another is called a joint. There are three main types of permanent joint and these may be classified as:—

FIG. 3. Longitudinal section of head of femur showing internal arrangement of the lamellae.

(a) fibrous
(b) secondary cartilaginous
(c) synovial

examples of which are shown in Fig. 4. The basis for this classification is the degree of movement possible between the two bones, but there are other characteristic differences which will be made clear in the following descriptions.

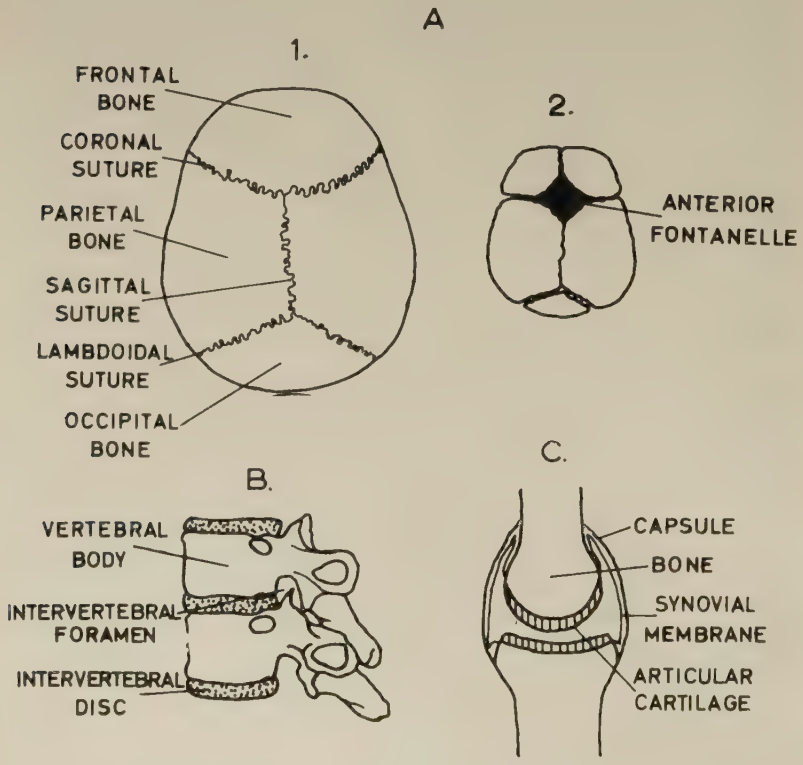

FIG. 4. Examples of the three main types of joint. A. Fibrous joint between bones of skull (1. adult: 2. infant), B. Secondary cartilaginous joint between vertebrae. C. Synovial joint.

Fibrous joints provide practically no movement between the bones involved. In this type of joint, the bony surfaces are bound together with fibrous tissue which may itself become invaded by bone in later life, allowing no movement at all. For example, the joints between adjacent bones of the

skull (which are called sutures) allow some slight movement during birth and some increase in skull size during childhood due partly to deposition of bone at the lines of bone junction. After about the age of thirty however, the fibrous tissue tends to become ossified.

Secondary cartilaginous joints are those bone junctions where the bones are joined together by cartilage and where some degree of movement is possible. The best example of this type of joint is the junction between adjacent vertebrae which form the skeletal framework for the neck and back. The joint surfaces of the vertebral bodies are covered with a translucent connective tissue called hyaline cartilage, and these are firmly united by a plate of more fibrous connective tissue called fibro-cartilage. This forms what is known as an intervertebral disc, the centre of which is much softer than the outer ring and is semi-fluid in consistency. Besides conferring on the vertebral column its appreciable mobility, the intervertebral discs function as shock absorbers reducing the effects of vibration transmitted along its length.

The third, and probably most important type of joint from the point of view of the athlete, is the synovial joint, which may allow considerable range of movement between the adjacent bones. The joint surfaces of the bones are covered with a smooth layer of hyaline cartilage, which contains no blood vessels or nerves but obtains its nourishment from the viscous liquid, called synovial fluid, which fills the intervening space. The synovial fluid is said to serve a lubricating function also and to provide for the removal of debris resulting from the wear of the joint surfaces. It is produced by the highly vascular synovial membrane which encloses it and extends from the margin of one joint surface to the other. Enclosing the whole joint is the fibrous joint capsule, which may be thickened in parts to form the intrinsic ligaments of the joint. These are composed of bands of collagen fibres running between the bones in directions which primarily serve to hold the bones in juxtaposition and to guide the direction of joint movement, by limiting the amount of movement in all other directions. Other bands of fibrous tissue, called the extrinsic ligaments, unite the bones at some distance from the capsule, but serve a similar function. In some joints there are intra-articular discs of fibro-cartilage interposed between the joint surfaces, dividing the joint cavity into two. Usually the disc is incomplete, allowing the two cavities to communicate with each other. The exact function of these intra-articular discs is not certain, but it is believed that they assist in distributing the synovial fluid more evenly between the articular surfaces.

There are six main classes of synovial joint:—

(a) ball and socket joints

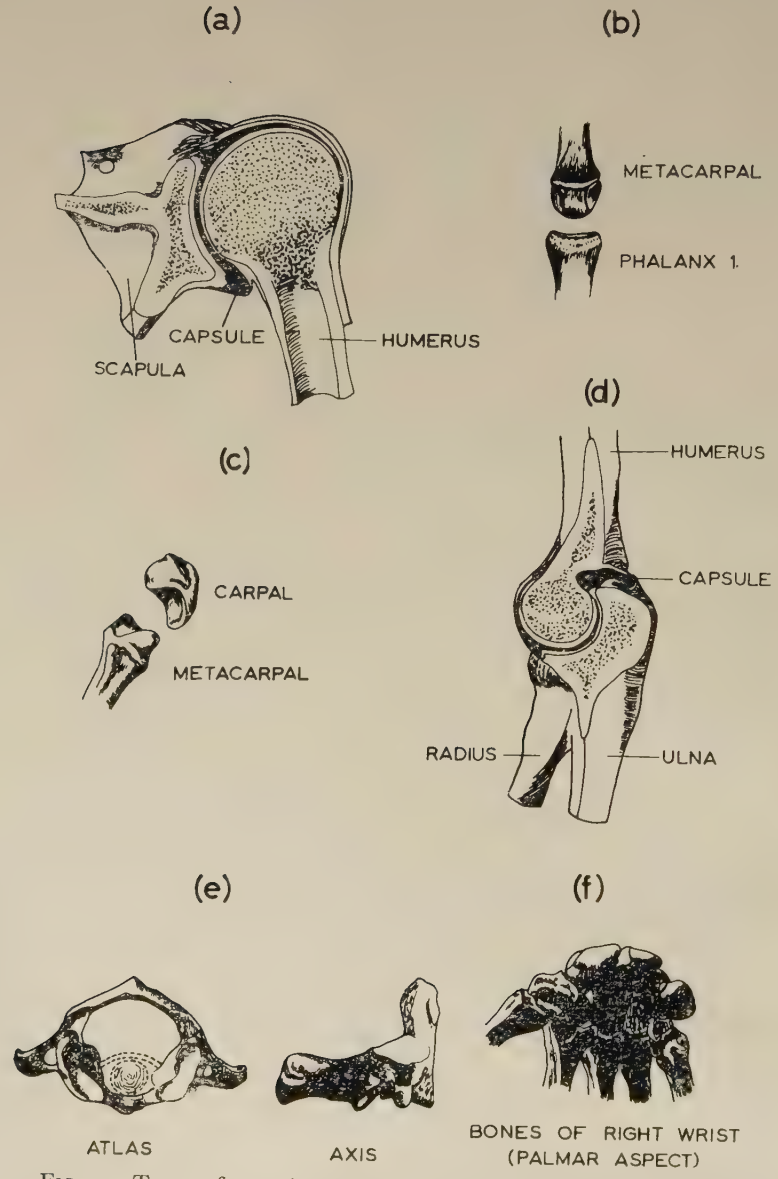

FIG. 5. Types of synovial joint: (a) ball and socket joint (shoulder): (b) condyloid joint (metacarpo-phalangeal): (c) saddle-shaped joint (carpo-metacarpal joint of thumb): (d) hinge joint (elbow): (e) pivot joint (atlanto-axial): (f) plane joint (wrist).

(b) condyloid joints
(c) saddle-shaped joints
(d) hinge joints
(e) pivot joints
(f) plane joints.

Examples of each are shown in Fig. 5.

The ball and socket joints permit free movement in all directions, i.e. flexion and extension (bending and straightening), abduction and adduction (outwards and inwards relative to the body), circumduction (a combination of all the previous movements) and rotation (twisting of the limb). One of the joint surfaces is spheroidal and fits into a cup of similar proportions and slightly greater dimensions. The best examples of ball and socket joints are the hip and shoulder.

Condyloid joints are modified ball and socket joints in which the ball and socket are ellipsoidal rather than spheroidal. They allow flexion and extension, abduction, adduction and circumduction but no rotation. The best examples are the wrist joints and the metacarpo-phalangeal joints at the base of the fingers.

Saddle-shaped joints permit freedom of movement in two planes; flexion and extension, and abduction and adduction. Each joint surface is saddle-shaped with one apparently inverted and placed at right angles to the other. The best example of this type of joint is the carpo-metacarpal joint of the thumb which is, however, capable of limited movements other than in the two main planes.

Hinge joints allow movement in one plane only; flexion and extension. One joint surface is drum or pulley-shaped and the other usually slightly cupped to conform approximately with the abutting surface. The best example is the elbow joint.

Pivot joints allow only one type of movement—rotation. There are two pivot joints in the body. The simpler of the two is that between the first and second vertebrae which is responsible for swivelling movements of the head, as in saying 'no'. The ring-shaped first cervical vertebra (called the atlas) carrying the skull, sits on a peg-shaped prominence from the second cervical vertebra (called the axis) and articulates with it in much the same way as the hinge of an iron gate.

Plane joints allow only a small amount of gliding movement. The joint surfaces are nearly flat and are bound to each other by inter-osseous ligaments. Examples of this type of joint are found at the junction of the short bones of the wrist or ankle.

The range of movement is therefore very dependent on the type of joint which exists between any two bones. However, there are several other factors which determine the range of movement at any particular joint. The strength and direction of the bands of ligaments at a joint have already been mentioned. In hinge joints such as the elbow, the capsule is thin in front and behind but much thicker and stronger at the sides, so that the joint surfaces are kept close together throughout the range of movement in the directions permitted, namely flexion and extension. There is similar restraint in the knee, which is a very much more complicated joint and one that is basically unstable because of the relatively poor fit of the articulating surfaces. However, the very powerful ligaments and muscles which cross the joint bind it together so successfully that dislocation of this joint is very rare.

Ligaments also limit the extent of movement in the directions permitted, for example in full extension of the knee or elbow joints: but in normal movements the limiting factor at most joints is muscular activity. This is particularly so in joints like the ball and socket shoulder joint where movement is possible in several different directions. Such an arrangement, in contrast to restraint by inelastic fibrous ligaments, has the advantage of keeping the joint 'tight' throughout its range of movement: but it has the disadvantage that a joint of this sort can be more easily dislocated by a sudden unexpected jolt. This risk varies with the form of the joint; for a deep socket almost enveloping a closely fitting ball, as in the hip joint, will provide a much greater safety margin.

Other factors which help to limit the range of movement at joints are limb girth and clothing. For example, the extent to which a man can flex his elbow or knee is often determined by the marked muscle development or the amount of fat in the area. There is a difference in the range of certain joint movements in men of different physique. In much the same way, bulky clothing restricts movements.

Muscles

There are three classes of muscle:—

(a) visceral muscle, which is found in the walls of the alimentary canal and blood vessels.

(b) cardiac muscle, present only in the walls of the heart.

(c) skeletal muscle, which is found in the limbs and trunk and is directly responsible for producing body movements.

Only skeletal muscle will be considered here.

Skeletal muscles are composed of bundles of muscle fibres (fig. 6). Each

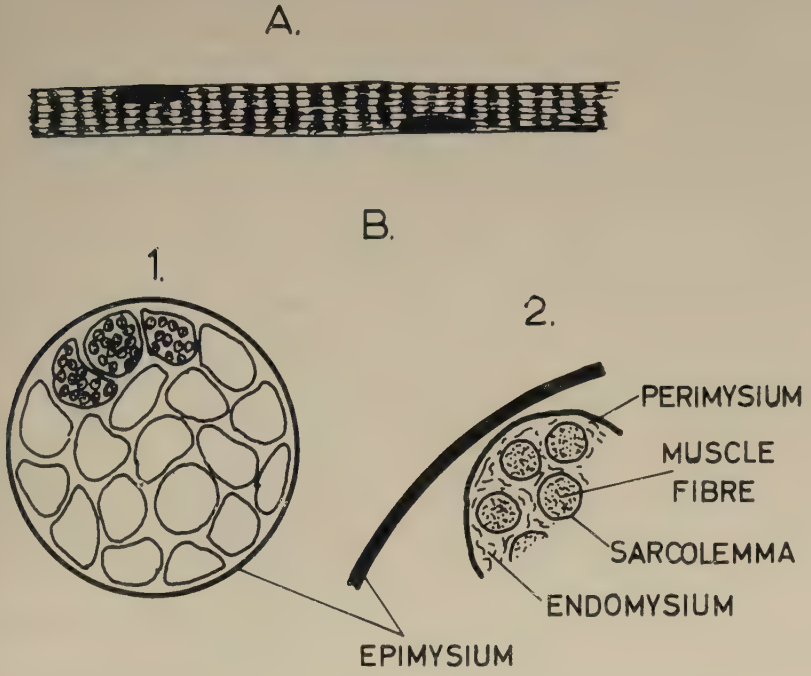

FIG. 6. A. Diagrammatic representation of somatic muscle fibre. B. Diagrammatic cross-section of a voluntary muscle. 1. shows a number of fasciculi within the epimysium: 2. is an enlarged view of one fasciculus.

fibre is a long, thin, cylindrical cell of contractile tissue which may vary in length from about 1 mm. to 5 cms., and occasionally may reach 15 cms. or more. The fibre is enclosed in a fine sheath of connective tissue called the sarcolemma. It is separated from other muscle fibres by a further layer of connective tissue called the endomysium. These fibres are gathered together into bundles, or fasciculi, which are bounded by a somewhat tougher membrane called the perimysium. The whole muscle is limited by a similar, and sometimes very much stronger, sheath called the epimysium. Generally speaking, there are fewer fibres in the fasciculi of muscles which are used for delicate movements than in those mainly employed in gross movements.

Most of the skeletal muscles are attached at one end to one bone and at the other end to another, so that contraction of their substance produces movement at the intermediate joint (Fig. 8A). This is not always so, however, and muscles may be attached to ligaments, fascia, tendons of other muscles and so

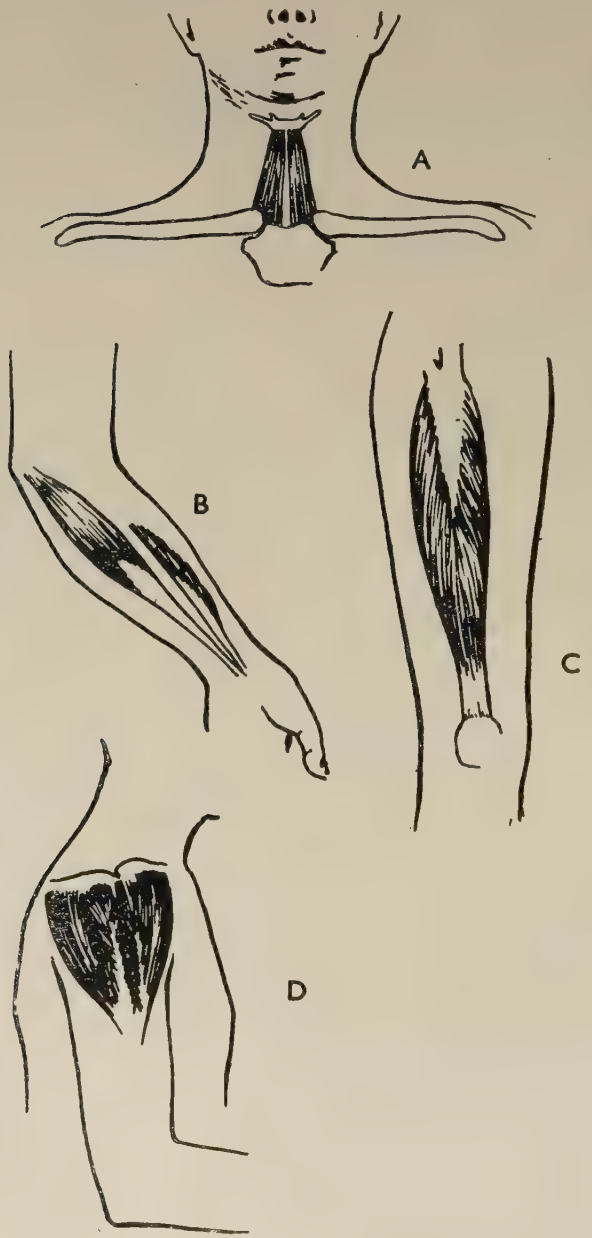

FIG. 7. Types of voluntary muscle. A. strap muscles (sterno-hyoids). B. a fusiform muscle (flexor carpi radialis) and a unipennate one (flexor pollicis longus). C. a bipennate muscle (rectus femoris). D. a multipennate muscle (deltoid).

on. Furthermore, two or more attachments of one end of a muscle may be to quite different structures; and if these are widely separated from one another, the muscle is said to have two or more heads. A good example of this is the triceps muscle, the upper end of which has three heads; the long head is attached at the shoulder joint to a part of the scapula (shoulder blade), the lateral and medial heads are attached to two different parts of the upper arm bone, the humerus.

At each end of a skeletal muscle the fibres run into collagenous tendon fibres through which they are attached to the bone or other tissue. Not all the fibres in a muscle are the same length; so that where the muscle gives way to tendon there is appreciable overlapping of muscle fibres with tendon fibres. The tendon of attachment may be long and thin, like a piece of cord, or broad and flat in the form of a sheet, called an aponeurosis. If the collagenous fibres are so short that they are invisible to the naked eye, the muscle is said to have a fleshy attachment. The muscle attachment which is relatively stationary during joint movement is called the origin, and the muscle attachment which moves is called the insertion. These terms are always relative to the movement produced, and in this respect a muscle may be likened to a spring arranged to close a door; the end fixed to the door frame would be called the origin and the end attached to the door the insertion.

The arrangement of the fibres is closely related to the functional properties of the muscle. There are two main patterns which may be found; one, in which the fibres are arranged parallel to the long axis of the muscle; another, in which the fibres run parallel to each other but are set obliquely to the length of the muscle. Examples of these are given in Fig. 7. Strap or 'fusiform' muscles have their fibres running directly from one point of attachment to the other, whereas the 'penniform' muscles have their fibres set obliquely into the tendon of attachment. Unipennate muscles have the tendon running down one side of the muscle only; bipennate muscles have the fibres attached to the tendon from two sides; multipennate types have a multiple attachment to a divided tendon.

The architecture of the muscle is significant, firstly because when a muscle fibre contracts it shortens to approximately half its length, and secondly, because the force of contraction is directly dependent on the number of fibres involved, irrespective of their length. Now when a muscle fibre contracts, it always does so to its fullest extent—it is incapable of partial contractions. Consequently, if two muscles which have the same volume are compared, the one which has a strap or fusiform arrangement of its fibres will be capable of producing a movement which is considerable in range but moderate in strength; and the one with a penniform, and particularly multipennate,

arrangement of its fibres will be capable of producing a strong movement over a restricted range.

The effective strength of a joint movement is also very closely related to the position of attachment of the muscle relative to the part of the bony frame-

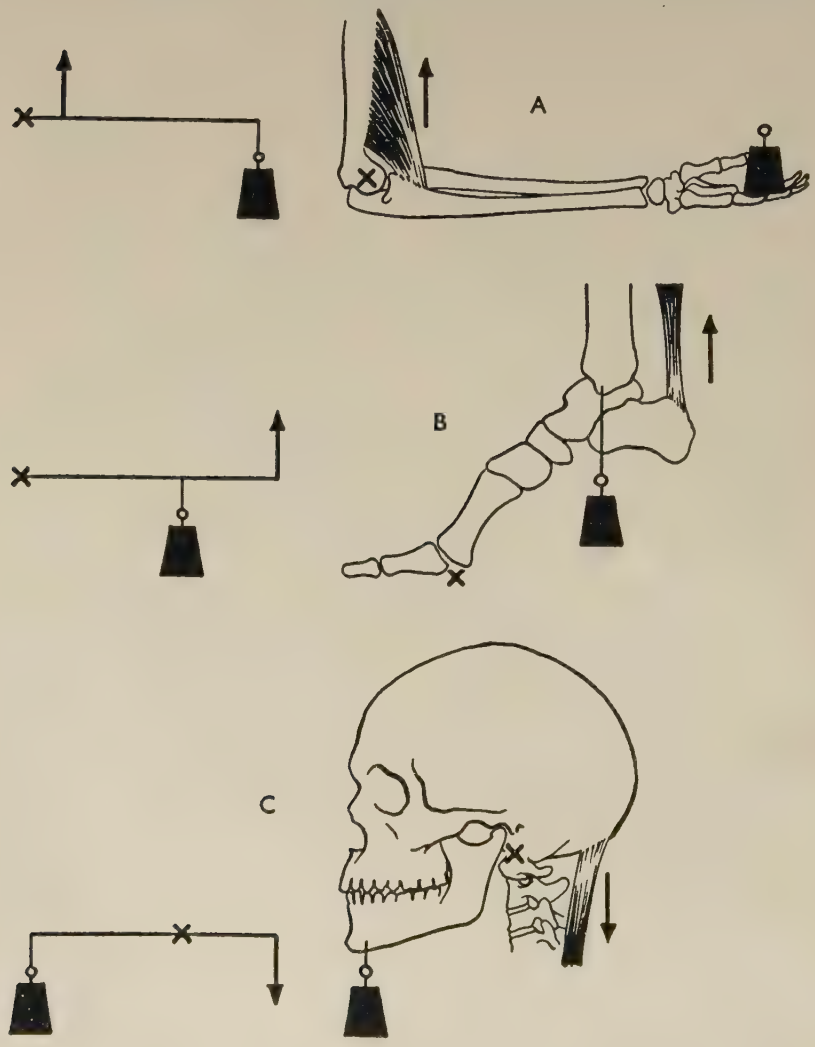

FIG. 8. The lever action of muscles. In each drawing the fulcrum is shown by a cross. A. the brachialis. B. the calf muscle. C. the semi-spinalis capitis.

work engaged in exerting or resisting forces in the environment, in other words, the mechanical advantage of the system. The three types of lever action which are found in the body are illustrated in Fig. 8; A. bending the elbow, B. raising the body on the toes and C. lifting the head. Clearly the greater the distance of the insertion of the muscle from the fulcrum of the movement, and the nearer to the fulcrum the point of application of the force, the greater the mechanical advantage. The effective distance of the muscle insertion from the joint or pivot varies with the limb position. A good practical example of this is found in the strength of flexion of the elbow, which is greatest when the elbow is bent to a right angle and the perpendicular distance of the insertion from the fulcrum is at its greatest. Furthermore (as anyone who has carried a heavy shopping basket knows), the effective strength of elbow flexion is greater if the resistance to movement (or weight) is applied somewhere along the forearm instead of in the hand.

However, in effecting a particular movement it is extremely doubtful if any muscle is ever active by itself. Limb and body movements require the co-operative contraction of many muscles both simultaneously and successively, and more will be said about this later.

The muscle primarily concerned in producing a particular movement is called the prime mover, and those which are concerned in assisting the movement are called synergists. The synergic muscle activity is essentially to prevent unwanted movement or to provide a firm base for the movement required. For example, flexing of the fingers would tend to flex the wrist as well unless the wrist extensors prevented it, while flexing of the elbow to a right angle, so that a glass of water could be held in the hand, would be useless unless the upper arm were maintained in a vertical position by muscular activity at the shoulder and many other parts of the body.

The muscles which may oppose a given action are called antagonists, and these are normally involved in providing a restraining or controlling influence on the desired movement. In a slow and deliberate movement, as the prime movers actively contract so the antagonists actively relax in order to steady the limb. In a swiftly executed action, the prime movers will initiate the movement but the antagonists will soon contract to slow it down or stop it; while a limb movement executed under the force of gravity, such as extending the elbow from a horizontal position, may be controlled solely by the antagonists of the action, in this instance, biceps and brachialis.

It has already been pointed out that when a muscle fibre contracts, it always does so to its fullest extent, i.e. its activity is 'all or none' in character. Consequently, when a muscle contracts to less than its maximal tension, some muscle fibres will be inactive. This forms the basic mechanism for the grading

of muscle contraction and joint movement—as more muscle fibres are activated, the greater the muscle tension and the stronger the applied force or the quicker the joint movement. However, the functional unit of muscle contraction is not the individual muscle fibre, but many muscle fibres served by the branches of a single motor nerve fibre, an arrangement called a 'motor unit'. Consequently, a nerve impulse which initiates a contraction of one muscle fibre will tend to produce contraction of all the other muscle fibres in the same motor unit. The number of muscle fibres served by one nerve fibre is known as the innervation ratio, which varies for different muscles of the body. The innervation ratio appears to be associated with gradation of muscle contraction; for muscles normally used for coarse movements have a high ratio while those known to be capable of extremely fine movements (such as the eye muscles) a very low one.

The component muscle fibres of a motor unit are not bundled together but are dispersed throughout a particular region of the muscle. It has frequently been suggested that, in a long-continued contraction of a whole muscle, as in postural activity, there is a rotation of motor unit activity, so that as some muscle fibres drop out from fatigue, others take over. No experimental evidence for this hypothesis has yet been obtained but it is an attractive idea.

The final limit to the continuation of muscle activity depends on the type of activity, but it is usually set by the failure of adequate blood supply to the muscle: for though the energy required by the muscle for contraction is derived from the breakdown of certain raw materials in the absence of oxygen, the reconstruction of these materials does require oxygen, and therefore an uninterrupted supply of arterial blood. Nevertheless, because of this mechanism, bursts of muscular activity may be undertaken in excess of the oxygen supply by running up an 'oxygen debt' which is paid off after the activity. This is a familiar experience to 100 or 220 yard sprinters, who do not become breathless until the race is over.

In the long-maintained muscle contractions of postural activity, the muscles may interfere with their own blood supply, because of the increase of intra-muscular pressure. Where the contractions are intermittent, the interruptions are intermittent and consequently less serious. In fact, intermittent contractions, especially of the lower limbs as in walking, can be beneficial because they help to pump the venous blood back to the heart.

Nerves

In the lowest forms of animal life, for example unicellular animals such as the amoeba, there is no nervous system and no need for one. If the animal meets some food, it engulfs it; for the whole animal comprises one cell.

Higher up the evolutionary scale, where animals have much larger bodies, certain cells have become specialised and may only be concerned with feeding

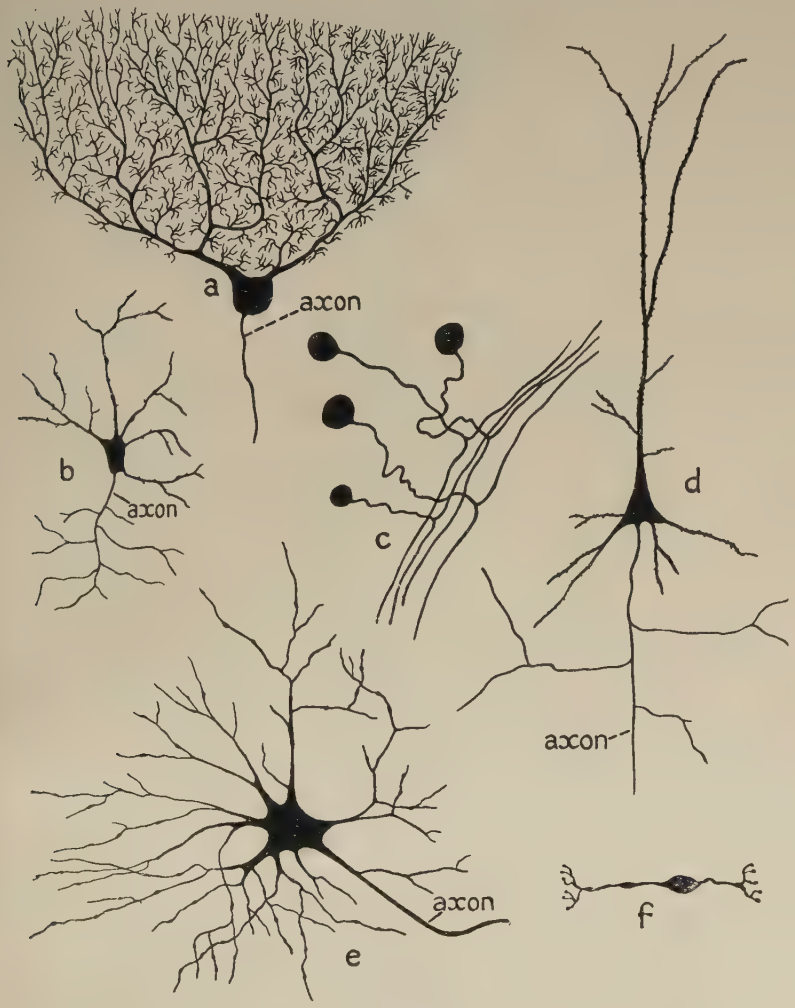

FIG. 9. Outline drawings of different types of nerve cells and their processes as they appear in Golgi preparations: (a) Purkinje cell from the cerebellum: (b) a short-axon nerve cell from the central nervous system: (c) spinal ganglion cells: (d) pyramidal cell from the cerebral cortex: (e) motor nerve cell from the ventral horn of grey matter in the spinal cord: (f) bipolar nerve cell from the retina of the eye.

or with body movements. To provide a rapid means of communication between these specialised parts of the body, other cells have become specialised in conveying messages. In the human body, this communication system has reached a considerable degree of development and provides an exceptionally complicated network of connections, far more intricate than any telephone exchange. The basic structural unit of this communication or 'nervous system' is the neurone. This consists of the nerve cell and its processes. As it is extremely small and difficult to see even under a microscope, most of what is known about its structure has been obtained by the use of special staining techniques to make it more visible. Different types of nerve cell (prepared by one of these staining methods) are shown in Fig. 9. Although the shape of the neurone varies in different parts of the nervous system, the basic structure is the same throughout. The simplest type comprises the nerve cell itself and two outgrowths or processes called nerve fibres. The one which brings messages to the nerve cell is called a dendrite, the one which takes messages from the cell is called an axon. An example of this arrangement which constitutes a bipolar cell, is shown in Fig. 9(f). A more usual type of neurone, where the cell has a single process dividing into a central and peripheral branch, is called a unipolar neurone, and it is typical of sensory or afferent nerve fibres. These fibres are so named because they are responsible for conveying from the periphery information concerning the current state of the skin, joints or muscles etc. which they innervate. The most common type of neurone found in the central nervous system has many dendrites and a single axon. The latter may be extremely long and extend down from its parent cell in the brain to the lower part of the spinal cord; or, if it is a motor nerve cell located in the spinal cord, from there to the end of the arm or leg. Such fibres

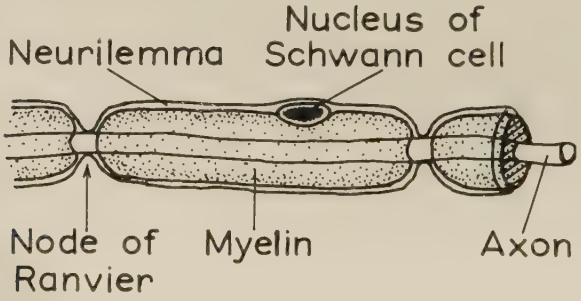

FIG. 10. Diagram of the structure of a nerve fibre. The axon is surrounded by myelin held in place by the neurilemma. Two nodes of Ranvier are shown.

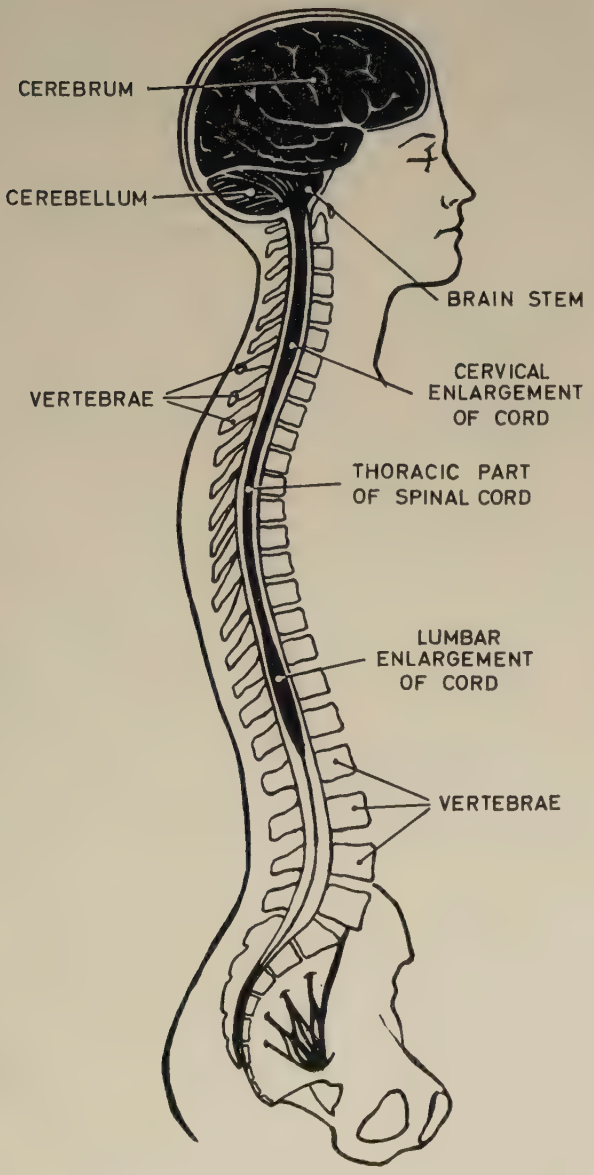

FIG. 11. The brain and spinal cord *in situ*.

have few branches and are termed motor or efferent neurones. Their function is to convey messages to the muscles of the limbs and so initiate movements.

The nerve fibre itself consists of an axon, of semi-fluid axoplasm, surrounded by a sheath of fatty white material called myelin, which is believed to provide effective insulation between nerve fibres and to keep their messages independent of one another (Fig. 10). In the central nervous system (that is, the brain and spinal cord—see Fig. 11) bundles of these nerve fibres, called tracts, provide the characteristic white matter. Where these fibres lose their visible myelin sheath and terminate on other nerve cell processes, the appearance changes to the familiar grey matter. Where nerve fibres leave the central nervous system and travel from or to the limbs and skin etc. in peripheral nerves, each fibre acquires a membranous sheath, called the neurilemma, composed of elongated (Schwann) cells. The thickness of the myelin sheath is generally related to the diameter of the nerve fibre—the thicker the axon, the thicker the myelin sheath. At intervals of about half a millimetre or so along the nerve fibre, the myelin sheath is interrupted at what are called nodes of Ranvier.

Messages are transmitted along nerve fibres by means of nerve impulses. Each nerve impulse produces a temporary change in the electrical properties of the surface membrane of the axon as it travels along the nerve fibre. The electrical disturbance can be picked up by suitable electrodes placed on the fibre and, after amplification, recorded on an oscilloscope screen or pen recorder. The electrical indication of the passage of a nerve impulse is called an action potential; and if a series of nerve impulses is transmitted along a nerve fibre, the size of the action potentials will be constant, providing the interval between them is not too short. But an action potential will only be propagated if the electrical disturbance detectable at the surface membrane has reached a certain critical threshold value. In other words, it has the same sort of "all or none" response as a muscle fibre. Immediately after the passage of a nerve impulse the nerve fibre is incapable of transmitting another impulse and is said to be refractory. This sets a definite limit to the rate at which impulses may succeed one another, and the speed at which impulses may travel depends on the fibre size. Impulses in large fibres travel at speeds of up to about 120 metres per second, but in very small nerve fibres the rate may be less than one metre per second. Because of these various limitations on the characteristics of nerve impulses, information can be transmitted only in terms of frequency of nerve impulses or their spatial arrangement, i.e. transmission along different fibres or pathways.

Although the structural unit of the nervous system is the neurone, the simplest functional unit is the reflex arc. The principal components are shown

in Fig. 12. Nerve impulses are initiated in a sensory receptor or end organ, which is made up of the peripheral terminations of a sensory nerve fibre specially modified to convert physical stimuli into nervous energy. These nerve impulses are then conveyed up the sensory nerve fibre, to enter the spinal cord through an intervertebral foramen, which is a small hole between adjacent vertebrae. The sensory nerve fibre enters the back of the spinal cord through the so-called dorsal roots and then branches to terminate on the dendrites of other neurones in the grey matter of the posterior horn (Fig. 12).

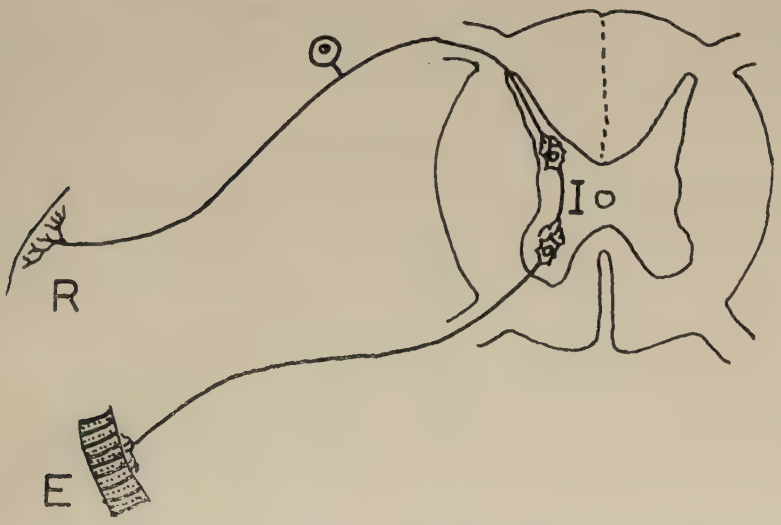

FIG. 12. Diagrammatic illustration of the simple reflex arc. R. is the receptor represented by free nerve endings in the skin: I. is an intercalated neurone and E. is the effector represented by a somatic muscle fibre.

The junction between the nerve endings of one neurone and another, called a synapse, allows nerve impulses to pass in one direction only; it acts like a non-return valve. Nerve impulses are therefore re-transmitted along a second or 'intercalated' neurone, which effects a synapse in the anterior horn of the spinal grey matter with one or more motor neurones. The axon of this cell emerges from the spinal cord through the ventral roots and conveys nerve impulses to an effector, such as a limb muscle, where an appropriate contraction is initiated through a number of motor units.

The simplest reflex arcs just involve a sensory neurone which synapsis directly with one or more motor neurones, but the only instance in which this

is known to occur is in the transmission of the stretch reflex, considered in more detail later. In all other reflex arcs, it is believed there is at least one intercalated neurone. At higher levels in the nervous system there may be several intermediate or internuncial fibres; and the greater the number of neural connections, the wider the range of possible responses: for at each nerve junction there is a possibility of using alternative pathways, and the number of synaptic connections possible in the brain is quite fantastic. It has been estimated that in the cerebral cortex alone, there are $6 \cdot 9 \times 10^9$ neurones with an average density of 10·5 neurones per 0·001 cubic mm.

The experimental work demonstrating the reflex activity of the nervous system is most easily appreciated by reference to the mechanisms operating in the maintenance of posture, which will be described in the next section.

POSTURE

Everyone knows that a sharp tap just below the knee of a person sitting with his legs crossed will produce a short kick or knee extension of the same leg. This is called a knee jerk and provides a demonstration of the basic mechanism of posture—the stretch reflex. The tap displaces the tendon of the knee extensor muscle (quadriceps femoris), with a consequent stretching of the muscle to which it is attached. In the muscle are certain sensory receptors which are sensitive to stretching, and these initiate nerve impulses which traverse the two-neurone reflex arc mentioned previously, and excite the main muscle fibres of the same muscle, causing it to contract and produce the kick.

In the maintenance of posture the same sort of mechanism is involved, except that the usual stimulus to stretching of the muscles is not a sharp tap applied to their tendon of attachment but gravity. In normal standing for example, the weight of the body constantly exerts a force vertically downwards and tends to flex the body at many of its joints. This flexing stretches the extensor muscles of the joints and reflexly produces contraction: but as soon as the receptors in the muscles are no longer stretched, no further impulses are transmitted through the reflex arc and contraction ceases. Consequently, whatever the postural position, a 'hunting' about the desired point occurs, in much the same way as any other elementary form of servomechanism, such as the thermostatic control of an electric oven. In its simplest form this involves a switch operated by a length of rod which expands or contracts with the heat. When the oven is switched on, it will gradually heat up and expand the rod until the desired temperature (corresponding to the thermostat setting) is reached. At this point, the rod has expanded

sufficiently to operate the thermostat control, which switches off the electricity and so allows the oven to cool down and the rod to contract. The contraction of the rod then operates the thermostat control and switches on the electricity again, to start another phase of the hunting cycle.

A schematic diagram of the main components of the stretch reflex is given in Fig. 13. The postural position is set by signals from the brain acting upon

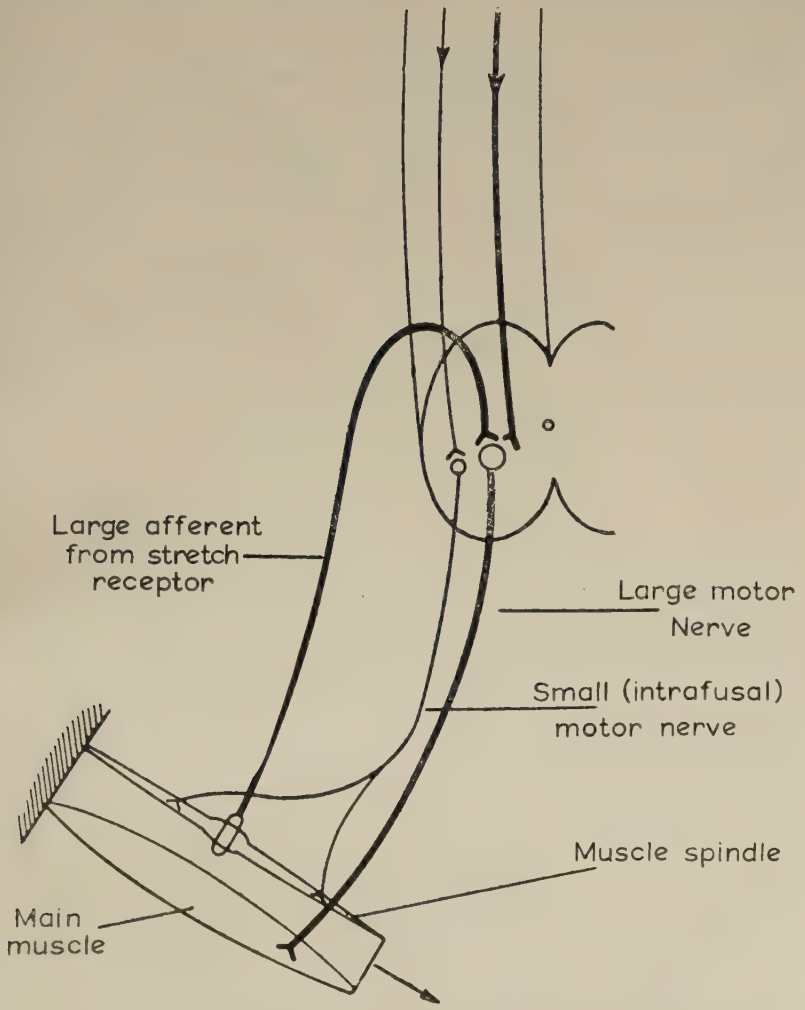

FIG. 13. Diagram of the basic elements of the stretch reflex system responsible for the servo-control of posture.

the small motor neurones, which send impulses to the contractile portions of the 'intrafusal' muscle fibres—specialised muscle fibres lying 'in parallel' with the main muscle fibres. Shortening of the contractile end portion of the intrafusal fibres then initiates impulses from the sensory nerve endings in the non-contractile central part to excite the main muscle fibres through the large afferents and their monosynaptic connection with the large motor neurones. This arrangement, which has been called a 'length servo', allows the muscle spindles (which are made up of groups or clusters of intrafusal fibres) to set the length of the muscle and consequently the joint position; for any external force which tends to stretch the muscle will reflexly cause it to contract and return the limb to its original position and so maintain the status quo. This system is only effective if it is continually working against some opposing force: otherwise a joint movement in the same direction as the muscle is pulling would be unopposed. In most instances, this opposing force is gravity; but the arrangement of antagonistic muscles enables the length servo to work on a reciprocal basis under any circumstances.

Afferent connections from the muscle spindles and other receptors in the limbs ascend in the spinal cord to a part of the brain called the cerebellum, which also sends efferent connections to the small motor neurones serving the intrafusal muscle fibres. Now the cerebellum not only receives information from the limb receptors (including skin) but from other sensory sources, including the organs of balance situated in the labyrinth of the internal ear. One of the main functions of the cerebellum, therefore, is the co-ordination of postural adjustments to maintain equilibrium of the body. It can be considered as providing a second level of integration of postural reflex activity, by relating information about the orientation of the head in space to the position of the rest of the body and initiating changes of attitude where appropriate.

There are three distinguishable organs of balance contained within the bony labyrinth of the inner ear. They are called the utricle, saccule and semicircular canals; and their relation to each other and to the cochlea of the organ of hearing are shown in Fig. 14. The utricle and saccule are known collectively as the otolith organs and each consists of a small cavity filled with a fluid called endolymph. Attached to the wall of the cavity is a small group of hairs, to the ends of which are attached some fine calcium granules which are acted upon by gravity. Depending upon the position of the head, therefore, the force of gravity causes deflection of the hairs and stimulates the hair cells to initiate appropriate nerve impulses. This mechanism is also susceptible, of course, to other influences such as linear acceleration or centrifugal force, and can therefore only indicate the resultant of such forces. The most convincing evidence which indicates the function of the otolith organs comes from

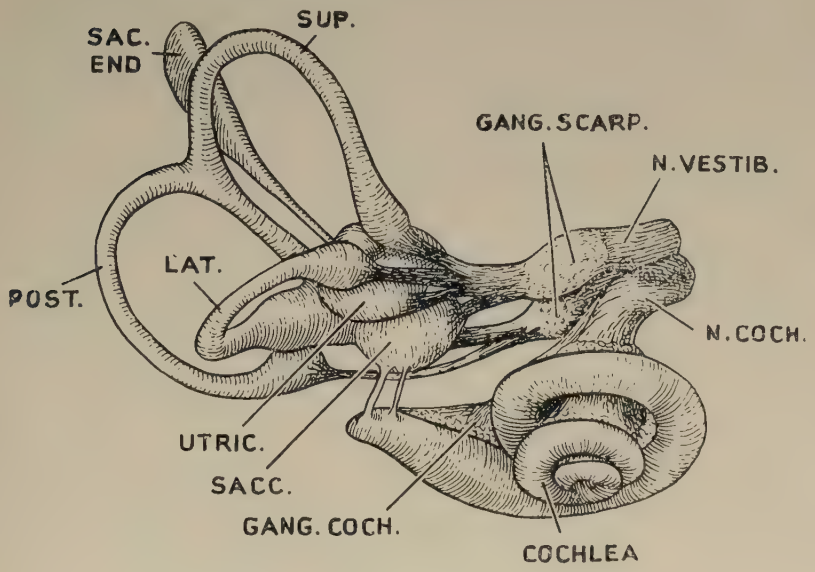

FIG. 14. Diagrammatic representation of the labyrinth. *N. Vestib.*= vestibular nerve: *N. Coch.*=cochlear nerve: *Gang. Scarp.*=Scarpa's (vestibular) ganglion: *Gang. Coch.*=cochlear ganglion: *Utric.*=utricle: *Sacc.*=saccule: *Sup.*, *Post.*, *Lat.*=superior, posterior, and lateral semicircular canals: *Sac. end.*=sacculus endolymphaticus.

experiments which have been carried out on lower animals, and particularly the prawn. This animal has an organ called the statocyst, which is very similar in structure to the utricle or saccule of man, except that, instead of fine calcium particles, the prawn uses sand grains, which it picks up from the sea bed. Every so often, the prawn casts off its cuticle or skin and at the same time sheds the lining of the statocyst. Prawns which had just moulted were kept in a tank where the sand grains had been replaced by iron filings and these were used by the animals for their statocysts. They were then subjected to the influences of a powerful magnet; and according to whether it was placed underneath, at the side, or above the tank, the prawns could be made to swim right-side up, on their sides or upside down.

The other organ of balance or equilibrium is composed of three semicircular canals, the planes of which are placed approximately at right angles to each other. At the end of each canal is an enlarged cavity, called the ampulla which contains a group of hair cells and calcium granules with much the same arrangement as in the utricle and saccule, except that the hairs are

believed to stretch almost completely across the canal. When the head is rotated, the walls of the canal move with the head, but the endolymph, through inertia, lags behind the movement and displaces the hairs. The hair cells are therefore stimulated by angular acceleration or deceleration of the head, but not constant rotation. In this way, the direction and rate of change of rotation of the head supplements the information derived from the otolith organs concerning linear acceleration and orientation under conditions of no movement.

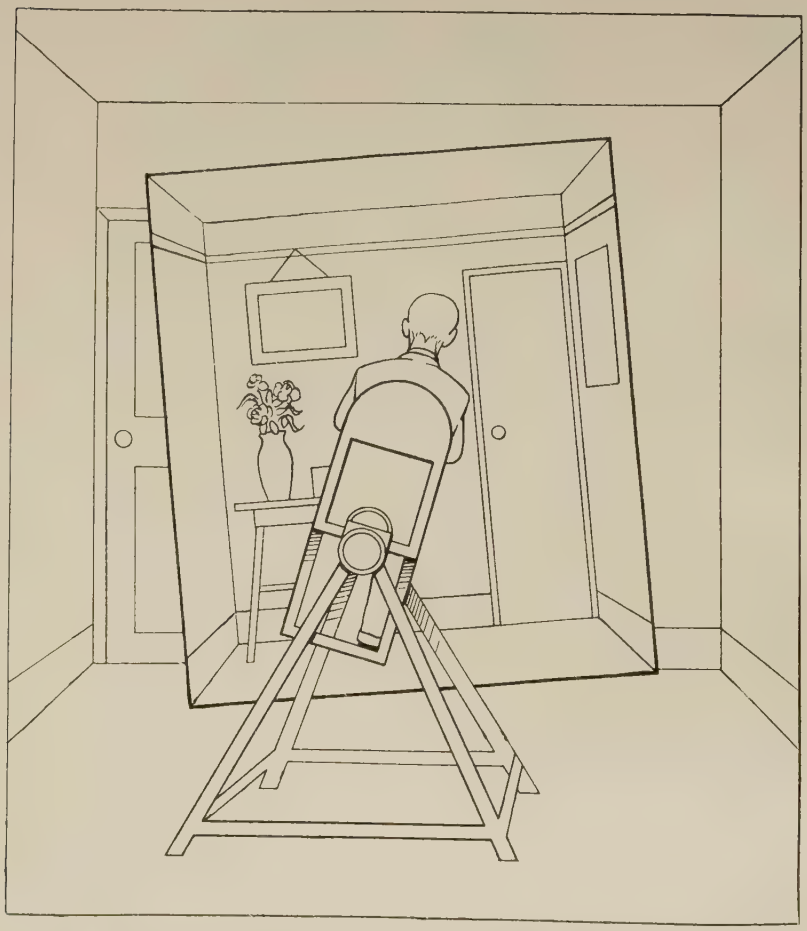

FIG. 15. Apparatus used for studying conflict between visual and labyrinthine indications of the position of the body in space.

Under normal circumstances in man, however, visual information appears to play the dominant role in the appreciation of position in space. This has been demonstrated experimentally by Witkin (1949) who seated a person in a tiltable chair which was placed inside an apparently ordinary furnished room. In fact, this experimental room could also be tilted at any angle, independently of any movement of the chair; and as the furnishings (table, picture etc.) were fixed rigidly to the floor and walls, no visual indication was given of the orientation of the room itself (Fig. 15). When the chair was tilted, the subject gained information about his body position not only from the labyrinthine receptors but also from pressure receptors in the skin etc. where the sides of the chair held him in place. Nevertheless, when both the room and the chair were tilted, many subjects either completely or partially ignored such impressions which were incompatible with the visual indication that the body was upright. When the room only was moved, there was induced in most people a strong illusion of body movement.

Situations which produce conflicting information from different sources concerning the orientation of the body are conducive to dizziness and nausea. In some of Witkin's experiments, which have just been described, certain subjects not only had great difficulty in making judgments but even became ill. It seems likely that the same conflict of sensory cues may at least be a contributory factor to seasickness. In other situations where the visual field is uniform or lacks detail such as when standing on the edge of a cliff or the roof of a high building, it seems probable that the feeling of insecurity or anxiety which most people experience may be partly due to the absence of normal visual cues.

MOVEMENT

All movements start from one posture and end in another. In many instances, of course, the overall body posture may be maintained constant, with local limb movements superimposed on it, such as in driving a car, riding a bicycle, or playing darts. In other circumstances, considerable body movement may be involved, particularly in gymnastics and athletic events, such as the high jump and pole vault or games such as tennis and squash.

It was remarked earlier in this chapter that 'all movements are initiated by nerve impulses which cause the contraction of muscles acting about joints'. This is a fundamental fact; but such a simple description can really only be applied to the almost random kicking and flinging of limbs characteristic of a baby in its cot. The movements made by people in the normal course of their daily lives are directed towards the achievement of some particular aim. Part

of the mechanism responsible for this organisation of movements has been considered in previous sections. At the third or highest level of integration, the movements of the whole body are co-ordinated to reach some desired objective and may be constantly monitored by information from various sensory sources in order to achieve a satisfactory performance. Such a function has commonly been ascribed to the cerebrum (see Fig. 11). The interrelationships between the cerebrum and the cerebellum in the control of movements and consequently of posture have been described by Walsh (1957) as follows: 'In balancing a ball on its nose the seal is supplied with visual and tactile information about movements of the object. It moves its head to keep the ball up, but the movements of the head involve readjustments in the trunk which may have to curve or lean over. At times the ball has to be allowed to fall because these adjustments have reached their limits, and if pushed further it would be the animal and not the ball that fell'. In other words, the achievement of some objective normally governs the posture adopted but when this has reached the limits of stability, it may indirectly promote a change of objective.

In balancing a ball on its nose, the seal is, in effect, faced with what is known as a 'tracking' task in the same way as a man driving a motor car or flying an aeroplane on a particular course. The aim is to keep the ball on the point of balance or the vehicle in the required direction, and any discrepancy between what is desired and the actual position at a given moment of time calls for a corrective movement to eliminate the discrepancy. By the nature of things, the correction may undershoot or overshoot, and a further discrepancy results which calls for another correction and so on.

In such tasks, a considerable degree of judgement may be required to determine the amount of the discrepancy and decide the extent and direction of the correction. In almost all ball games (for example, soccer, rugger, cricket, tennis, baseball etc.) certain aspects of the play make demands of judgement concerning the direction and speed of the ball and the estimated position in which the player should be placed to achieve interception. Unfortunately, it is impossible to consider these perceptual problems in detail here.

In making the initial movement, or subsequent corrective movements, in most sports or games of skill, there are two particularly important features: (1) the spatial and temporal organisation of the muscle contractions involved and (2) the timing of the movement as a whole relative to the situation. Concerning (1), the importance of this factor depends on the complexity of the movement in terms of the number of muscles and joints involved. For example, to make a particular stroke with the racquet or bat in tennis or cricket requires the contraction of a large number of muscles, not only in a given

FIG. 16. Stroboscopic photograph of the service in lawn tennis.

order but at very critical times relative to one another. Some indication of this is given in Fig. 16 which is a stroboscopic photograph of the service in lawn tennis. The different amounts and directions of the movements of various parts of the body in such a rapid manoeuvre indicate the critical timing in-

volved. The second point mentioned refers to the timing of the movement as a whole relative to the target. For example, the player in Fig. 16 is making the service stroke at the anticipated position of the ball, which must be hit at a certain phase of the movement sequence if the shot is to be successful. Similarly, in cricket or golf, the player must make his particular stroke so that the bat or club hits the ball at the appropriate point in the movement cycle.

These two characteristic features are not only true of bat and racquet games but also of kicking and throwing. A complicated movement sequence which must be related to some external object is part of practically every activity demanding physical skill. Field athletics, (for example discus, hammer, javelin etc.), bowling in cricket or pitching in baseball all have as their basis a complicated movement sequence leading to the release of the missile at the appropriate phase. Kicking in football or punching in boxing pose similar problems. Jumping activities, such as the high jump, pole vault, gymnastic and high diving or springboard events also demand extraordinarily complicated movement patterns directed to crossing an obstacle or achieving a particular body position at some definite phase of the movement cycle.

That people vary in their ability to make the complicated movement patterns demanded of a particular sport is a matter of common observation. No doubt much of this individual variation is due to the different amounts and types of training, and more will be said about this later. The odd thing is that very little is known about how or why people differ in motor ability when they have had the same degree of training. In fact, it is not even known if it is justifiable to talk about motor ability as a term for some generalised aptitude for making highly complicated movements successfully. There are, and have been, many well known 'good all-rounders', but whether these folk are exceptionally gifted or whether most people who excel at one sport may also excel at certain others given the appropriate training is again not known at the present time. As such questions are also influenced to some degree by other facets of human behaviour, such as personality and motivation, it is unlikely that any quick answers will be found.

The effects of training on the ability to make movements are much better known. The flailing movements of the baby in its cot have already been mentioned as an example of an undirected and relatively unorganised series of muscle contractions. Purposeful movements, however, are learned, and there is experimental evidence to show that the first stage of developing control over a particular movement is the elimination of unwanted elements. A movement which is at first produced only as part of some gross activity is gradually isolated as one learns to relax the muscles which contribute nothing to the desired movement. This is true not only of control of muscles responsible

for unusual movements, such as moving the ears or toes, but also of the extremely complex movement sequences of a golf or tennis stroke. It is a common observation that the novice at such games appears to be extremely tense, trying to contract almost every muscle in his body at the same time!

The second stage of gaining control over a movement seems to be the contraction of the right muscles at the right time. It is, however, impossible to say in any detail which is the right time for a given muscle to contract in a particular movement sequence—whether it is an action with the hand, a kick with the foot or a jump with the leg. People vary considerably in the pattern of their movement sequences (i.e. style) and what may be suitable for one person, through reasons of body build and previous training etc., may be unsuitable for another. The main change which appears to take place during training of a skilled movement is in the consistency of the timing of the muscle contractions relative to one another. This makes the whole movement sequence more predictable or reliable and consequently it can be related to its external target or objective with much more chance of success. The importance of learning a particular movement sequence is seen when it is realised that corrections can only be made intermittently. The delay in responding to a perceived error is known as the 'reaction time'. This varies appreciably from one situation to another according to the number of alternative courses of action available. If the choice lies between one of two conditions, the time taken to respond will only be about a fifth of a second, but in most everyday situations there are several alternatives, and the reaction time may be as long as half a second or even more. Consequently, the numerous muscle contractions of movements which are executed in times less than this must be learned as a complete sequence and initiated without the possibility of correction or modification. In other words, to achieve the minimum requirements for successful execution of rapid or relatively complex movements, the pattern of the constituent muscle contractions must be learned in its entirety. But the better any given movement is learned (i.e. the more consistently the various muscle contractions are made), the longer or more complicated the movement sequence may be before a particular level of error is reached, or a correction need be made. And the more reliable or predictable the movement, the more accurately it can be matched to its objective, and the more skilled or accomplished is the overall performance.

REFERENCES

Chambers, E. G. (1946). *Statistical Calculation for Beginners.* Cambridge: University Press.

Evans, F. G. (1957). *Stress and strain in bones.* Springfield, Illinois: Thomas.

Walsh, E. G. (1957). *Physiology of the Nervous System.* London: Longmans Green.

Witkin, H. A. (1949). Perception of body position and of the position of the visual field. *Psychol. Mon.* 63, No. 302.

SUPPLEMENTARY BIBLIOGRAPHY

Clark, W. E. Le Gros (1958). *The Tissues of the Body.* 4th Edn. Oxford: Clarendon Press.

Morehouse, L. E. & Miller, A. T. (1959). *Physiology of Exercise.* 3rd Edition. St. Louis: Mosby.

Sinclair, D. (1961). *An Introduction to Functional Anatomy.* 2nd edition Oxford: Blackwell.

2

PHYSIOLOGICAL FACTORS LIMITING MAXIMUM PERFORMANCE EFFECTS OF TRAINING

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PHYSIOLOGY AND PSYCHOLOGY

In a healthy man whose body is functioning normally, exercise performance is seldom limited by the overloading of a single organ: rather the stresses which arise in strenuous exercise are widely distributed, so that breakdown represents inadequacy over the whole range of physiological function. The point when this occurs depends on the man's reactions both to the imposed physiological strain and to the psychological stress to which this gives rise. The latter is determined by his constitution and motivation, but also by the extent to which he has exposed himself to comparable stresses during training or previous events. It is widely believed, for example, that knowledge of his ability to continue exercise on a treadmill beyond the normal breaking point by changing over to oxygen breathing, was one of the factors which spurred on Roger Bannister to complete his first four minute mile. Complete self-mastery may enable the subject to drive himself into unconsciousness, as did Peters during the marathon at the 1954 Commonwealth Games at Vancouver, but such single-mindedness is rare.

PHYSIOLOGICAL SUB-DIVISION OF ATHLETIC EVENTS

From the physiological viewpoint, athletic events can be sub-divided into three main groups. First, those of such short duration that the energy of muscular contraction can be provided by anaerobic metabolism within the muscles without the need for additional oxygen from the blood. In these events muscle strength and neuromuscular co-ordination are of prime importance. Sprinting and many field events come into this category. Second, events of longer duration in which ability to deliver oxygen to the active tissues is of paramount importance. The quarter-mile, which is just too long to be sustained by anaerobic metabolism alone, falls into this category, of

which the mile is perhaps the supreme example, but two and three miles should also be included. Third, distance events in which the ability to cope with monotony and discomfort and with the heat liberated by prolonged physical exertion are primary considerations.

MUSCLE STRENGTH

(a) *Constitution*

Muscle strength is the characteristic feature of sprinters and wrestlers. Those who excel in these activities tend to be of the muscular or mesomorphic type, with broad shoulders and narrow hips, big muscles and heavy bones. With this constitutional advantage such subjects are likely to respond to programmes of muscle training to a greater extent than their more asthenic or rotund companions, but the differences are only ones of degree.

(b) *Training*

Increasing strength due to training is accompanied by an increase in the size of individual muscle fibres, in the thickness and strength of the sarcolemma and intramuscular connective tissue and in the firmness of their attachments to the bones. These changes are consequences of prolonged use under near maximal conditions but there is still controversy on the minimal requirements necessary for their attainment. In this context Muller and his co-workers (Muller 1958)[1] have claimed that the exertion of only one-third of the power of a muscle in a single short isometric contraction once a day is all that is required. The findings, which are so far uncorroborated, take no account of the need for appropriate development of the blood supply within the muscle and of the general circulation to sustain the hypertrophied muscles. This aspect needs to be investigated in relation to the effects of the training procedure on endurance as well as strength before this attractive method of muscle development can be advocated with confidence.

In addition to strengthening the muscles, training leads to increased economy of effort so that the same amount of useful work is performed with a smaller total expenditure of energy. This has been demonstrated in wheelbarrow pushing (Crowden 1928),[2] bicycling (Gemmill *et al* 1930),[3] uphill treadmill walking (Knehr *et al* 1942)[4] and also walking on the horizontal treadmill (Cotes and Meads 1959).[5] The improvement is believed to be due to better co-ordination and direction of movement rather than to any increase in the efficiency of muscular contraction.

The effect of muscle training can only be conferred by the repeated active participation of the muscles concerned. It is not enough, for example, to train

intermittently or to train the circulation or the contralateral muscles. Thus Kruse and Mathews (1958)[6] found that when they exercised the flexor muscles of the left elbow in a group of students there was no effect on the muscle strength of the contralateral muscle group: a gain in strength occurred on the same side provided that the exercise was performed three to five times per week: solitary or twice weekly exercise was not sufficient as a means of increasing strength, though it might be sufficient to maintain it once the training process is complete. Equally, a high state of cardiovascular efficiency conferred by endurance training does not imply a high level of skeletal muscle strength. On the contrary, a relatively low level of strength may be associated with exceptional performance (Cureton 1956).[7]

When training stops there is a decline in muscle strength and in associated muscle size. The rate of deterioration appears to be slower than that of acquisition and to leave behind a 'memory' of the training process which enables the muscles to be re-trained more rapidly on a subsequent occasion. This is also true of training of the heart and circulation. The effect on exercise ability is illustrated in Fig. 1 (Fletcher, 1956),[8] which shows the time a subject could step on and off a 22 inch bench at a frequency of thirty per minute on consecutive days. Exercise ability increased slowly during the first

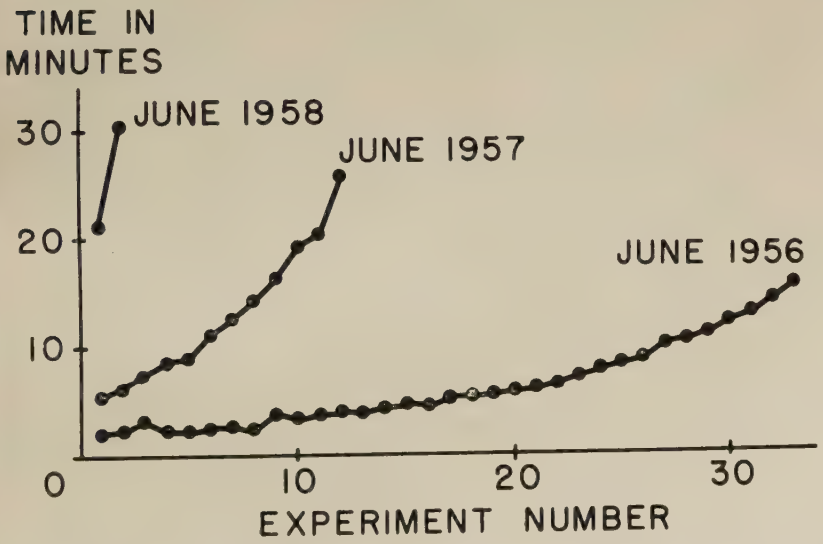

FIG. 1. Effect of daily repetition of stepping exercise upon the time for which it can be maintained. Step height 22 in., step frequency 30 per minute. (Fletcher; 1960a, by kind permission of *J. appl. Physiol.*).

series of experiments, then fell off when exercise was discontinued. A year later, however, it was still better than on the first occasion and the rate of improvement with training was greater. These changes in exercise ability were more marked the following year.

The inactivity of bed rest further decreases exercise ability and there is associated loss of muscle tissue, bone calcium, muscle strength and cardiac circulatory efficiency. When the patient starts movement again recovery follows the same order. Thus in a group of six subjects studied after a three to four week period of complete bed rest, H. L. Taylor and his colleagues (1949)[9] found that muscle strength and co-ordination returned within a few days. Cardiac efficiency returned in two to five weeks, but it was seven weeks before the subjects regained their previous cardiovascular responses to a manoeuvre in which they were tilted from the horizontal to the semi-vertical position. Similar changes occurred in one of the subjects when he was submitted to a surgical operation which kept him immobile for three weeks.

OXYGEN TRANSPORT

The ability to transfer oxygen from the air to the muscles is exceptionally well-developed in performers in middle distance events; such men are typically long and lean and only moderately muscular. Their long legs enable them to cover the ground quickly with minimal dispersion of energy in up and down movement (Fenn 1930),[10] (Cotes and Meade 1960)[11] and their relatively light build reduces the load they carry. It has still to be determined whether their build is also associated with a high respiratory or cardiovascular potential. It is possible, for example, that their lightly-built chest cage is a constitutional advantage, since a minimal amount of energy is needed to move it during the vigorous ventilation of strenuous exercise. In this respect such subjects are likely to differ from their more substantial companions.

The transfer of oxygen, with which we are concerned, is from the air to the muscle fibres: this must be regarded as one functional process although it embraces the whole range of respiratory, cardiovascular and circulatory function and the respiratory function of the blood. Each of these is important because the ability of the system as a whole depends on the oxygen carrying capacity of its weakest link. However, because the components have developed and operate as a unit, they are closely integrated and share the load imposed by strenuous exercise. Thus, although limitation of the heart's output is usually the prime factor determining the tissue oxygen supply, the actual breaking point may be determined as much by respiratory as cardiovascular considerations.

(a) *The Respiratory Apparatus*

We can consider the respiratory apparatus under a number of headings. First as a pump or bellows which moves air in and out of the lung; second as a gas mixing chamber in which inspired gas is distributed to the different groups of alveoli: third, as an exchange mechanism where oxygen and carbon dioxide pass in opposite directions between the gas in the alveoli and the blood in the capillaries which line their walls. The effectiveness of the mechanism depends on the properties of the membrane separating the gas from the blood phases; these include its area, thickness and diffusion constant and the volume of blood with which the gas comes in contact. Finally the lungs and chest cage together act as a sensory organ which, when overstimulated, gives rise to the symptom breathlessness. This sensation, however caused, is a major factor limiting maximal exercise performance.

These different aspects of lung function are discussed below. They are considered in greater detail in *The Lung* by Comroe[12] and his collaborators in Philadelphia, which can be recommended to those who require fuller information.

(i) *The Bellows Function of the Lung.* The bellows function of the lungs is best considered in terms of the maximum breathing capacity (M.B.C.). This is the maximum amount of air which the subject can shift per minute when breathing as hard and as fast as possible. The average value in normal young men, aged twenty, is about 160 litres per minute, but there is a big individual variation. This is in part explicable in terms of trunk length, which is highly correlated with total height, and of muscle strength; but other unidentified factors also contribute. Increase in maximum breathing capacity with training of the muscles of respiration might be expected to occur, but it has not been conclusively demonstrated. The maximum breathing capacity of middle-distance runners is not exceptional although they use their respiratory muscles very actively during periods of strenuous exercises. On the other hand, research workers, who perform the maximum breathing capacity test repeatedly on themselves, often have high values and the same may be true of mountaineers. Dr Pugh, who made this measurement on himself at intervals throughout the 1953 British Mount Everest Expedition (Pugh 1958),[13] found that his M.B.C. was increased on return to sea-level as compared with his initial value. During the period at great altitude he also recorded high values, but this can be explained in part by the decreased air density at altitude. Both the research worker performing the M.B.C. and the high altitude mountaineer undertaking strenuous exercise in a rarified atmosphere, are ventilating at nearer to their maximum than the athlete competing at sea level, and this

may account for their better ventilatory performance. Indirect corroborative evidence for this hypothesis is obtained from the vital capacity, which is the maximum amount of air a subject can expire after a forced inspiration: this measurement correlates fairly well with the maximum breathing capacity. Available measurements suggest that high values may be obtained in wrestlers, weight lifters and oarsmen, in whom the upper shoulder girdle is exceptionally developed. In the first two instances, at least, such subjects have no special need for extra reserves of ventilatory power.

The bellows function of the lungs varies with age. The maximum breathing capacity rises during the period of growth, reaches a peak at about the age of twenty, then falls on average by 1-1½ litres of ventilation per minute for each year of life. The M.B.C. is limited mainly by the resistance to flow offered by the air passages in the lungs: this varies with the degree of lung inflation, being least when the chest is fully expanded and infinite when the airways are closed at the end of a forced expiration. The way in which this process is affected by age is considered further below. It is also affected by smoking, recurrent bronchitis and other chest illnesses. Active respiratory effort, as in glass blowing, and the playing of wind instruments, has also been incriminated, but, as in other spheres, there is no good evidence that strenuous activity is in any way harmful to the healthy organ.

(ii) *Alveolar Capillary Function.* The uniformity of gas distribution within the lung is also adversely affected by increasing age, but in young subjects it is almost perfect; thus no consequences of training or of exceptional fitness in this respect are likely to be detectable. By contrast, variations may occur in the pulmonary diffusing capacity, which is the rate of oxygen or carbon monoxide transfer across the alveolar capillary membrane (e.g. Bannister *et al*, 1960).[14] High values for this index, which has attracted much attention amongst physiologists, appear to be essential to athletes if they are to attain very high levels of energy expenditure (Shepard, 1958).[15] How this facility is acquired is still unknown. It may be a consequence of exceptional permeability of the membrane or enlargement of alveolar surface due to anatomical variation; these would be hereditarily determined and if combined with large lungs and long limbs, would constitute the human equivalent of the greyhound. Alternatively, as a result of increased blood flowing during strenuous exercise the alveolar surface may become surrounded by more capillaries and hence there may be more blood in contact with the alveolar gas. Further investigation of these alternatives are under way in laboratories throughout the world and we shall doubtless soon have a better understanding of this aspect of respiratory physiology.

(iii) *The Control of Respiration.* Ventilation rises during exercise to make

available increased quantities of oxygen to the blood and to wash out carbon dioxide from the lungs. At most levels of activity the ventilation is proportional to the energy expenditure (Fig. 2). This is achieved through a com-

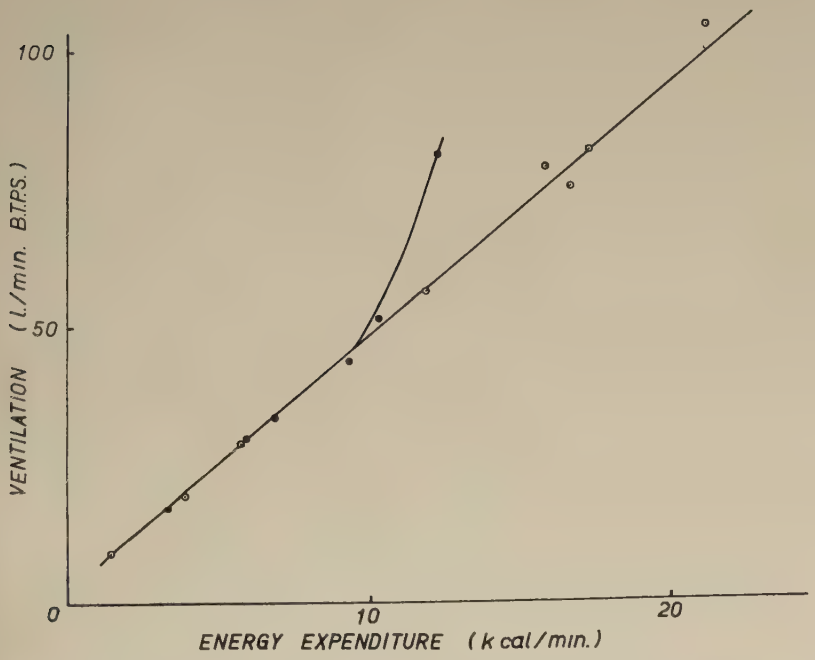

FIG. 2. Comparison of ventilation on exercise between M.H., a middle-distance runner (open circles) and a subject having a lower maximum exercise ability (closed circles).

bination of stimuli acting on the respiratory centre in the brain, which controls the muscles of respiration. The setting of this control mechanism may be considered in terms of the ventilation equivalent for oxygen, which is the ventilation per 100 ml. of oxygen consumption or the slope of the line in Fig. 2. The ventilation equivalent varies from one subject to another and is high in patients with breathlessness due to heart or lung disease. It is widely believed that low values occur in athletes and that the ventilation equivalent falls as a result of training. This may be so, but no change occurred in a group of army recruits studied over a three month period of physical training (Cotes and Meade 1959),[5] and the author has no reference to a change occurring on moderate exercise in practised subjects on whom serial measurements have

been made. During near maximal exercise, on the other hand, such changes undoubtedly occur: the ventilation increases disproportionately, due in part, to the stimulus to respiration which arises from the liberation into the blood of lactic acid produced in the active muscles. This change occurs at a higher level of energy expenditure in athletes than in subjects who are out of training or who have a lower capacity for exercise. These differences are illustrated in Fig. 2, which shows the relationship between ventilation and energy expenditure for Martin Hyman, a middle-distance runner, and the author, who is not athletic. The ventilation equivalents of the two subjects are identical up to an energy expenditure of 10 kcal/min; at higher work levels the relationship for the athlete continues unchanged whereas in the non-athlete the ventilation increases abruptly. This difference, which affects the relative abilities of the two subjects to increase their muscle blood supply, rather than the effectiveness of their lungs, is considered below.

(b) *Cardiovascular Function*

The passage of oxygen across the alveolar capillary membrane from the alveolar gas to the blood in the alveolar capillaries leads us from consideration of the respiratory apparatus to a discussion of the heart and circulation and the respiratory properties of the blood. The effectiveness of oxygen transfer depends on the quality of the heart as a pump, on the blood as an oxygen carrier, and on the relative calibre of the blood vessels throughout the body, including the active muscles.

(1) *The Heart.* The heart of the athlete differs from that of the average subject in a number of ways (e.g. Asmussen and Neilsen, 1955;[16] Astrand, 1956;[17] Rushmer and Smith, 1959[18]; Karpovitch, 1959).[19] At rest the cardiac outputs are comparable but the athlete has a low pulse frequency with a correspondingly increased stroke volume. This combination leads to economy of cardiac effort so that the resting work of the heart is somewhat reduced (Mellerowicz, 1956).[20] On this account athletic training, and, to a lesser extent, regular physical exercise, has a beneficial effect which offsets the strain of maximal activity. Athletes can take comfort from Rook's findings (1954)[21] that life expectation in Cambridge University Blues is at least as good as in other graduates: and mortality experience amongst Finnish skiers (Karvonen, 1959)[22] and United States college athletes (Montoye *et al*, 1956)[23] is equally encouraging.

During exercise, also, the pulse frequency for a given level of energy expenditure is lower in athletes than in others (e.g. Fig. 3). At the same time the stroke volume is greater so that the total cardiac output is high. Much of the blood goes to the active muscles where the high rate of blood flow has the

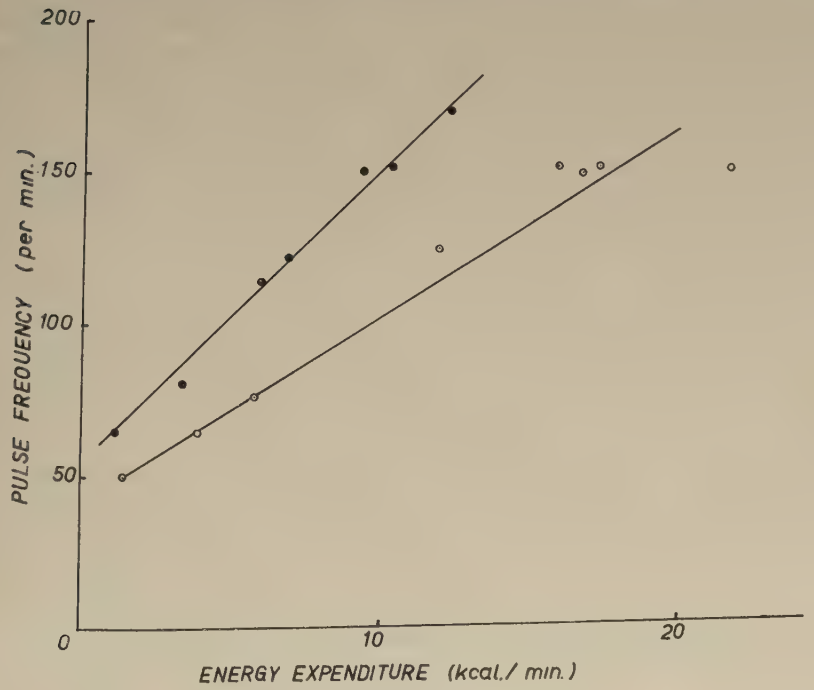

FIG. 3. Comparison of exercise pulse frequencies between M.H., a middle-distance runner (open circles) and a subject having a lower maximum exercise ability (closed circles).

effect of reducing the amount of oxygen removed per unit of circulating blood: in this way the venous and tissue oxygen tensions are kept up and there is less need for anaerobic metabolism, by which oxygen is obtained from pyruvate at the expense of lactic acid production. The level of lactic acid in the blood and the oxygen debt incurred at the end of exercise are correspondingly less. A relative lack of oxygen is most likely to occur in the muscles at the start of exercise before the blood supply has adjusted itself to the new level of activity. It is therefore natural that the rate of increase of cardiac output, as gauged by the pulse frequency, rises abruptly in the athlete at the start of exercise: in others it rises more gradually under conditions which impose the same degree of cardiac strain as judged by the final exercise pulse frequency (Fig. 4). The athlete's pulse frequency also returns more rapidly to its resting level after exercise because there is a smaller oxygen debt to be repaid.

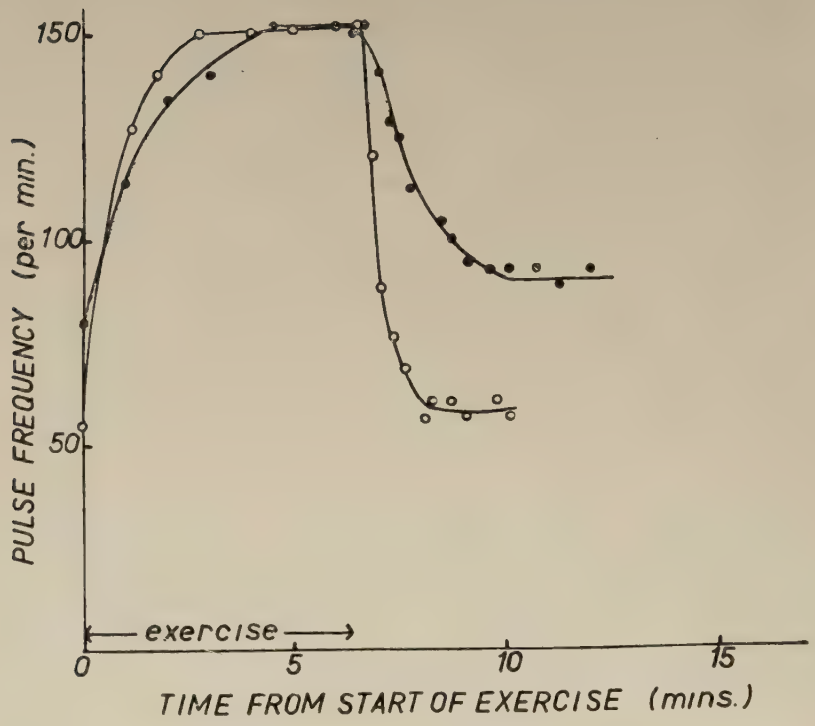

FIG. 4. Pulse frequencies over periods of exercise of comparable severity (pulse frequency 150 per min.) for M.H., a middle-distance runner (open circles) and a subject having a lower maximum exercise ability (closed circles). Exercise walking at $5\frac{1}{2}$ m.p.h. and 4 m.p.h. on inclines 1 in 12; energy expenditures 16.6 and 9.3 kcal/min. respectively.

During maximum exercise, the pulse frequency often exceeds 200 beats per minute, and virtually all the oxygen is removed from the blood on its circulation round the body. In those who are not athletic this occurs at a high pulse frequency and a relatively low stroke volume; in the athlete the heart rate is slower even during maximal exertion. Stroke volume remains high, so that each ventricle of the heart may be ejecting per beat more than its own total weight in blood. The maximum exercise ability is increased in proportion.

(ii) *Training of the Heart and Circulation.* The ability to increase cardiac output is partly hereditary: it may also be acquired by training. Training is very effective as any sedentary person can discover for himself by recording his pulse rate at the end of a one-minute period of stepping exercise before and

after a strenuous fortnight's holiday. A bench 20 inches or 22 inches high and a step rate of thirty times per minute are often used for this type of assessment in athletic young men (Brouha *et al*, 1943;[24] Fletcher, 1960).[25] A step height of 16 inches or less and a lower frequency is more suitable for adolescents, women and older subjects, Ryhming (1953)[26] and Taylor (1944)[27] used a treadmill, Wahlung (1948)[28] a bicycle ergometer. There are three main ways to record the pulse frequency:—(a) during recovery after a fixed period of exercise, which is usually one or five minutes; (b) during exercise of predetermined severity; (c) during exercise of increasing severity; the purpose is to get the maximum exercise pulse frequency and energy expenditure. The last is the best measure of exercise ability but it takes much time, exceptional co-operation from the subject, an experienced investigator and appropriate technical facilities. If these are available, maximal exercise ability can be determined, expressed either as maximum work rate in watts, horsepower, kilogram metres or foot pounds per minute; or in terms of the maximum attainable oxygen consumption (the aerobic capacity) or cardiac output. More often the maximum exercise ability is estimated indirectly by the use of the linear relationship which exists between pulse frequency and energy expenditure (Fig. 3). The pulse frequency and energy expenditure are independently determined at two levels of sub-maximal exercise and the energy expenditure at an arbitrarily chosen maximum pulse frequency, usually 170 or 180 per minute, is then found by extrapolation; for example in Fig. 3 the maximum energy expenditures of the two subjects are approximately 22 and 13 kcals/min. This method is only approximate, since the relationship is linear only to within about 10 per cent of the aerobic capacity. If one neglects this factor, the maximum oxygen intake is under-estimated by about 1·5 kcal/min. (Wyndham *et al*, 1959).[29] The maximum pulse frequency attainable on exercise varies from one subject to another and decreases progressively with age (Astrand *et al*, 1959,[30] Robinson, 1938).[31]

Training which can be detected by these tests of physical fitness as well as by athletic prowess, affects the right side of the heart from which blood is expelled into the pulmonary artery and the lungs, as well as the left side, which supplies the rest of the body. There is accompanying hypertrophy of the cardiac muscle, but much of the increase in heart size evident on radiological examination of the chest is a result of dilatation and an increase in the volume of blood in the ventricles during the relaxation phase of the cardiac cycle. During the subsequent contraction, relatively more of the blood is expelled than in the untrained, so that the end contraction volume of the heart is reduced.

The severity and duration of exercise necessary to attain a useful increase

in cardiac efficiency has still to be ascertained. Maximal development, as judged by a greatly reduced pulse frequency during exercise, appears to be closely related to endurance training, such as is undertaken by long-distance runners, in whom the work load of the heart is increased regularly for long periods. Weight-lifters, sprinters, and competitors in other short distance events have significantly higher values (Fig. 5, Fletcher, 1960).[25] At the same

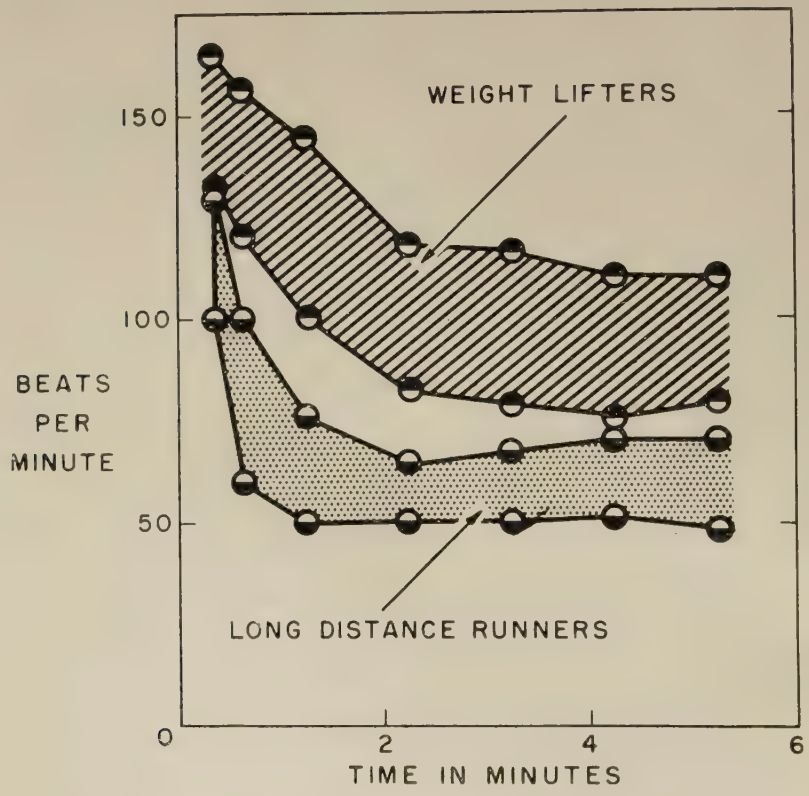

FIG. 5. Effect of endurance training on pulse recovery curves. Pulse frequency during recovery from a one minute period of exercise, stepping thirty times off a 22 in. bench, for weight-lifters and long-distance runners. (Fletcher; 1956b, by kind permission of *Ergonomics*).

time it has been observed (Karvonen, 1959)[22] that tennis players, provided they perform regularly, have greater cardiac efficiency than heavy manual workers whose average level of energy expenditure is vastly greater. Similarly,

Fletcher has shown on untrained subjects that short duration exercise has an effect. The explanation appears to lie in the extent of the pulse rate increase during exercise. Karvonen found in a series of experiments in which untrained medical students ran on a treadmill half-an-hour daily for four weeks that it was only when the pulse rate was increased to approximately 60 per cent of the maximal possible increase (i.e. to a frequency of about 150 per min) that a marked training effect was obtained.

(iii) *The Circulation.* During athletic training, there are changes not only in the heart but throughout the systemic vascular bed. It seems probable there are also changes in the pulmonary bed. The capillaries of the heart and active muscles profilerate and there is an increase in the sensitivity of the mechanisms controlling the distribution of blood to the different parts of the body. These are directed both to increasing the venous return and to re-distributing the cardiac output, so that an increased minute volume of blood is available for the active muscles and for the skin, where it is required for heat dispersion. The increased flow of blood to these regions is due to vaso-dilatation and therefore the blood pressure of an athlete during exercise may be lower than that of an untrained man. There is a compensatory adjustment of blood flow away from the abdominal viscera and inactive regions of the body: the blood flow to the brain is apparently unaffected. These important adaptations need more study. Their effects are evident in the rapid increase in cardiac output at the start of strenuous exercise and in the increased tolerance to prolonged exertion which is acquired by training. Over-compensation may occur, leading, for example, to excessive diversion of the blood away from the kidneys and intestines; and this may be a factor contributing to the haematuria and diarrhoea which occurs in marathon runners. These appear to be transient phenomena and no long term consequences have been reported.

The volume of blood in circulation and the total amount of haemoglobin in the body both increase with training. The haemoglobin percentage and hence the oxygen carrying capacity per 100 ml. of circulating blood remain at the normal level; polycythemia does not occur except in those who live at high altitudes. The red corpuscles in the blood are unaffected by training except that their average fragility is reduced; probably because the less robust cells are prematurely fragmented by the high blood flow velocities and rate of flow reversal during strenuous exercise. This destruction may occur suddenly when someone who is out of condition takes severe exercise and it may cause temporary anaemia and reduction in exercise tolerance.

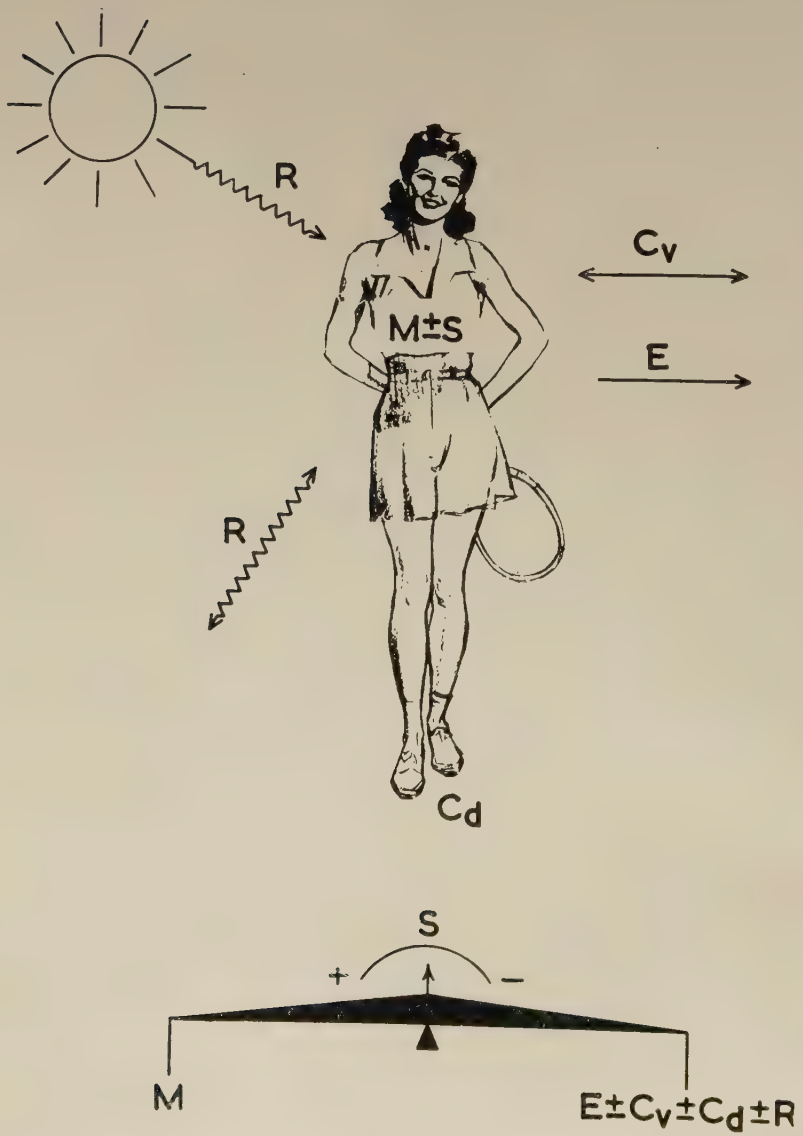

FIG. 6. The avenues of heat exchange with the environment, showing the contribution of metabolism (M), body storage of heat (S), evaporation (E), convection (Cv), conduction (Cd), and radiation (R). (Fox, 1960, by kind permission of *Ergonomics*).

BODY TEMPERATURE CONTROL

a. *Normal Mechanism*

The human body is a fairly efficient convertor of chemical into mechanical energy. Nevertheless only one-third of the energy liberated is so utilised: the remainder needs to be dissipated as heat. If the period of exercise is short or the exercise intermittent, this is of little consequence, since the body's capacity to store heat serves as a buffer to smooth out any abrupt rise in temperature. The heat eliminating mechanism can dispose of the surplus heat at leisure. If the exercise is strenuous and lasts more than half an hour, the body must lose the surplus heat as it is produced or incur the consequences of an excessive rise in internal body temperature. The calorifuse mechanisms are illustrated in Fig. 6 (Fox, 1960).[32] Heat is exchanged between the skin and its surroundings by convection (Cv.), conduction (Cd.) and radiation (R.). It is also dissipated by evaporation (E.) of water from sweat, except in a very humid environment. The exchange depends on the temperature gradients, the area of contact and thermal characteristics of the surroundings, and the radiant heat load; the evaporative heat loss depends on how much sweat the man produces and the characteristics of the environment, that is to say the ambient temperature, humidity and wind velocity. There is some evaporation also in the lungs. Heat storage (S.) serves as a buffer between metabolism (M.) and the means of heat disposal.

During exercise the temperature gradient between the skin and its surroundings is increased by dilatation of skin blood vessels and increased blood flow. These changes tend to increase the outward flow of heat. However, heat loss by this route only plays a limited part in heat dispersion under normal conditions; and in a hot environment the flow may be reversed, so that the body temperature rises. Under these circumstances, heat dispersion is due entirely to evaporation.

This entails diversion to the skin of blood which might have gone to the active muscles and the loss as sweat of fluid which would otherwise have sustained the blood volume and hence the output of the heart. Together they throw a considerable strain upon the circulation which is reduced by heat acclimatisation.

b. *Heat Acclimatisation*

The load upon the circulation is greatest in long distance events when heat dispersal must somehow keep pace with heat production if an incapacitating rise in body temperature is to be avoided. Training of the circulation and acclimatisation to heat, therefore, are complementary in such events. Both

processes contribute to increased cardiac output and expansion of blood volume. At the same time endurance training probably increases heat tolerance and heat acclimatisation is likely to assist endurance training: but there is indirect evidence that an interaction of this sort is relatively rare. Heat acclimatisation plays no part in formal athletic training, except perhaps in Finland, and even there it probably contributes little to the production of distance runners. On the other hand, instances of thermal collapse do occur and the performance of distance runners in this country is sometimes impaired on a hot day. At the 1958 British Commonwealth Games in Cardiff, when the arena temperature was 25°C., the British runners in the six miles event returned times well below their best. Coincidence cannot be excluded, but a possible connection was demonstrated by laboratory investigations carried out soon afterwards: it was then found (Fig. 7. Bannister and Cotes,

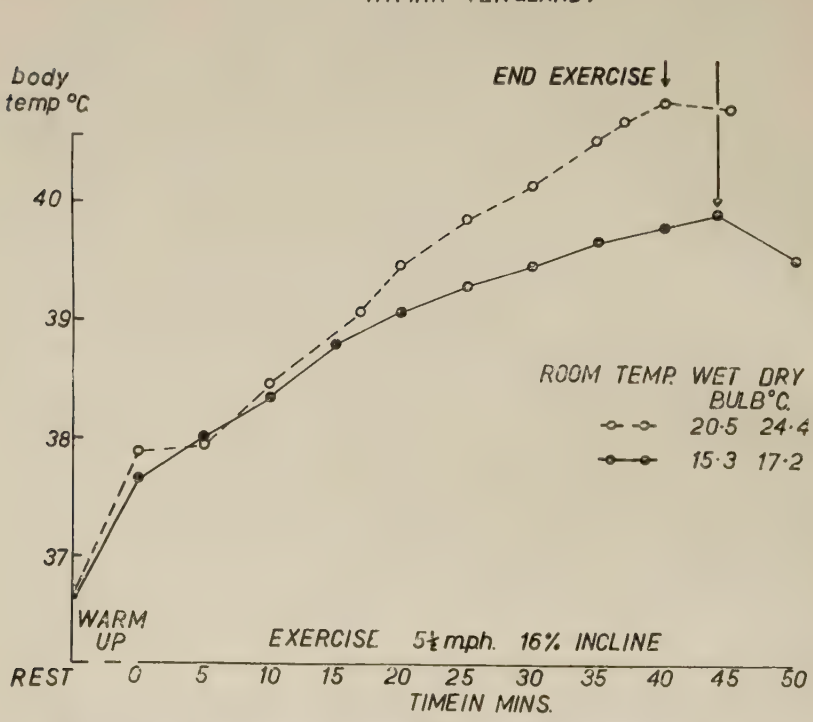

FIG. 7. Body temperature changes during strenuous exercise of the same severity at two levels of ambient temperature in subject M.H. (Bannister & Cotes; 1959).

1959)[33] that body temperatures in these athletes were higher and their work performances lower when exercising at 25°C. than at 15°C. By contrast, two United States athletes who were heat acclimatised, were found by Robinson to maintain their performance at similar high temperatures. These studies suggest that men living in temperate climates, though they benefit because their normal environmental temperature is suitable for endurance training, may be at a disadvantage in competitions held in warmer climates. This thermal handicap can be overcome by heat acclimatisation, but an appropriate procedure has still to be perfected. During the week before embarkation the athletes might benefit from graded intermittent daily exposure in a hot room equipped for exercise; or they might acclimatise by arriving early at the competition site.

c. *Cold Acclimatisation*

(i) *Cold Air.* Excessive heat loss due to cold is a rare hazard in athletes since the heat generated by activity is usually enough to offset it. Where this is not so, as in mountaineering or polar exploration, properly designed clothing and equipment can reduce the heat loss (Newburgh, 1949).[34] This is a characteristically human adaptation. It is supplemented by physiological and psychological acclimatisation to cold over a period of days. The extent and effectiveness of this adaptation is still under investigation.

(ii) *Cold Water*

a. *Anatomical Considerations.* Physiological mechanisms give limited protection against cold water immersion because heat is lost by conduction from the body. This is a serious hazard. Loss of heat affects first those with little internal thermal insulation in the form of subcutaneous fat. Women are

FIG. 8. Competitors in the 1951 Cross Channel Race (by kind permission of the *Daily Mail*).

better insulated than men, but individual variation is enormous. It is only those with a large constitutional fat component who are able to maintain their body temperature for long periods of immersion, and can therefore participate effectively in distance events such as cross channel races (Fig. 8). The relatively constant body temperature of one of these subjects during swimming and cold water immersion is shown in Fig. 9 along with those of a lean subject, whose body temperature fell precipitously. (Pugh and Edholm, 1955).[35]

b. *Physiological Aspects.* During normal exercise body temperature rises.

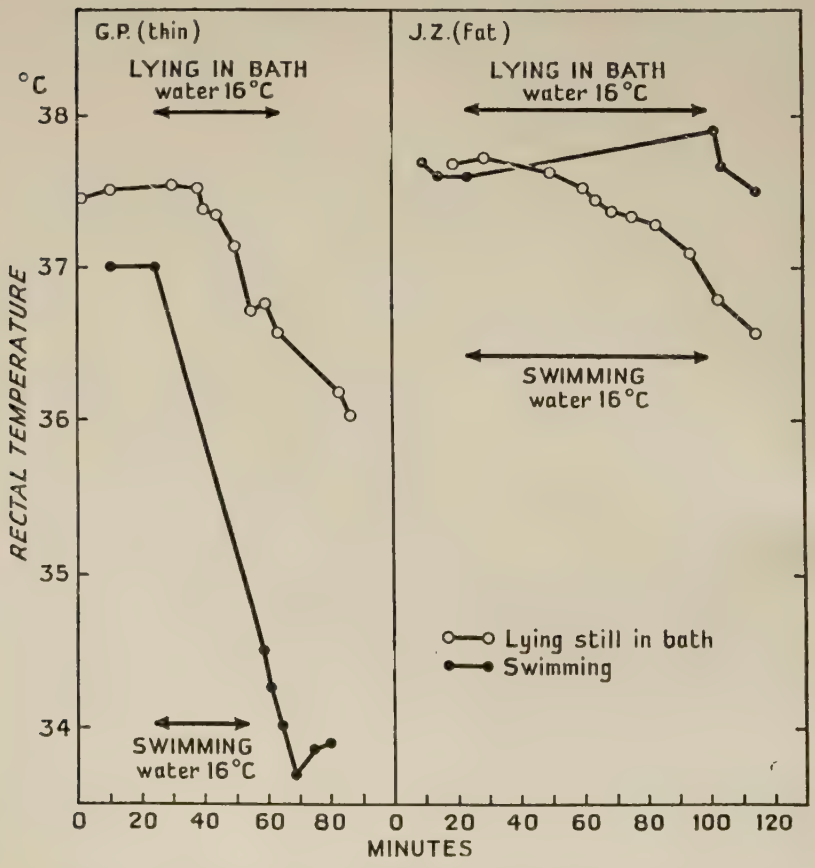

FIG. 9. Rectal temperatures after swimming and during immersion without swimming in water at 16°C in G.P. (thin) and J.C. (Fat). (Pugh and Edholm; 1955, by kind permission of *The Lancet*).

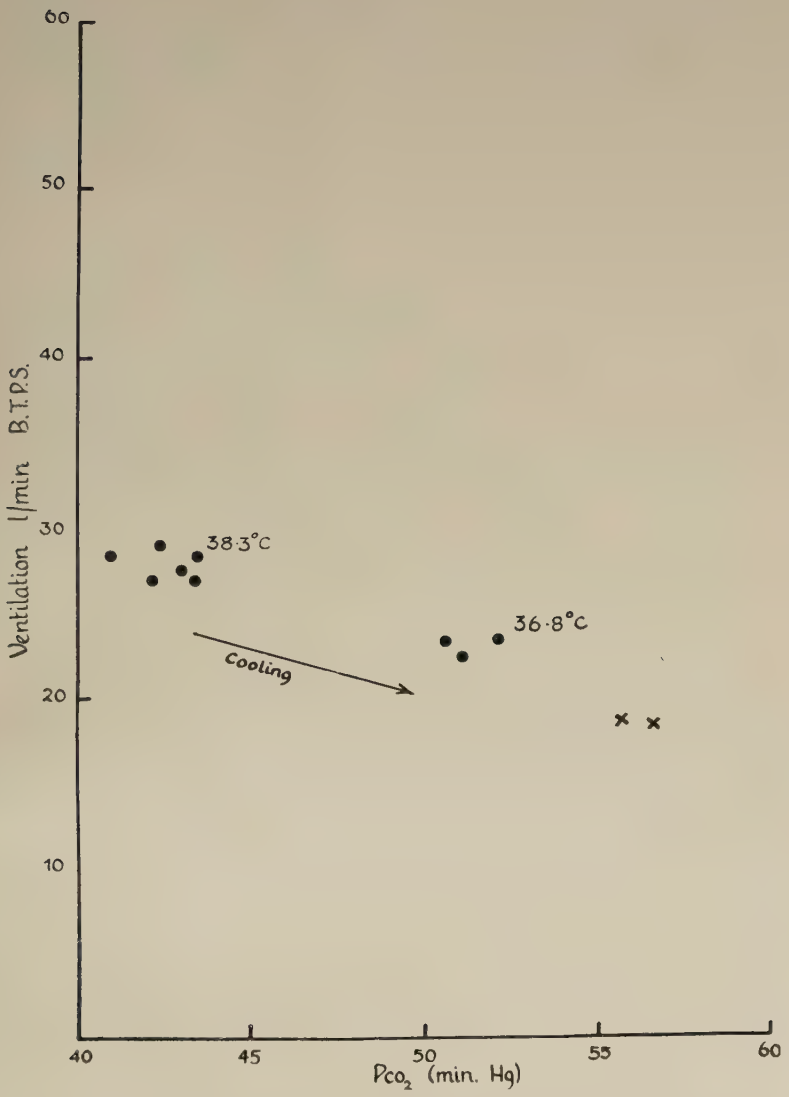

FIG. 10. Effect of body cooling during steady state exercise on body temperature, ventilation and end tidal carbon dioxide tension (P_{CO_2}) for a subject breathing oxygen. Closed circles refer to steady body temperature, crosses to conditions when the temperature was falling. (Cotes: 1955).

This is a disadvantage when the rise is more than moderate because of the strain which is thrown upon the circulation and ultimately upon tissue function—especially that of the central nervous system. In moderation it is a physiological mechanism contributing to increased respiration, cardiac output and efficiency of muscular contraction. If the body temperature during exercise rises less than normal, or fails to rise as a result of immersion, function is imparied even though no actual reduction in body temperature takes place. For example when a man breathes oxygen under these circumstances his ventilation may rise less than normal: in consequence he excretes less carbon dioxide from the lungs and the blood carbon dioxide tension rises. This is illustrated in Fig. 10. The carbon dioxide retention is disadvantageous because the gas is a vasodilator; the rise in blood concentration secondary to hypoventilation may therefore increase skin blood flow and hence the rate of fall of body temperature. A vicious spiral of increasing physiological derangement is then set up by further depression of ventilation. Unless the spiral is broken by shivering or rescue, the blood carbon dioxide concentration will rise to a narcotic level. This is the more likely to occur when there are other unfavourable factors, such as exhaustion or an unserviceable underwater oxygen breathing set.

Men vary in their ability to tolerate carbon dioxide and the assessment of this tolerance has been used as a guide in the selection of underwater swimmers and divers. Plump people are often unusually tolerant; and this attribute, as much as the buoyancy and thermal insulation which their fat depots provide, may contribute to their performance in a number of aquatic events. The buoyancy of adolescence, which is a factor in the exceptional performances of young people over short distances, is probably not associated with any comparable tolerance to carbon dioxide; but the possibility bears further investigation.

d. *Warm-up.* The improvement of exercise performance when body temperature rises seems adequate justification for a general warm-up before an event. This serves a different purpose from the formal repetition of a manoeuvre requiring special skill and co-ordination in order to ensure that the pattern is right. In practice the latter is almost certainly of value: whether the former is much use is open to question. It may be that its greatest value is in allaying anxiety rather than as a means of promoting physiological efficiency. This is suggested by the very individualistic warm-up procedures adopted by some athletes and the disregard of it by others who have broken records whilst 'running cold'. Over short distances Karpovich and Hale (Karpovich, 1959)[19] have demonstrated that running times are not affected by warm-up, and their work has not been effectively refuted. In long distance

events, warm-up may be a psychological aid, a point not yet investigated; but its tendency to raise body temperature is not always an advantage. In events where there is a risk of hyperthermia it is better to start cool rather than hot.

Many athletes regard warm-up as a prophylactic against injury, on the grounds that warm muscles are less liable to strain. This may be so, especially where skilful and complicated movements are concerned: but whether the advantages are due to practice of the movement pattern or to a rise in body temperature has not been established.

FACTORS LIMITING MAXIMUM PERFORMANCE

a. *Normal subjects: Comparison with effects of heart and lung*

We have seen how, in events requiring maximal exertion for more than about half a minute, the cardiovascular and respiratory apparatus are the main limiting factors. The strain thrown upon the individual links in the chain of oxygen transport varies from one type of exercise to another. In middle distance races, it is widely believed, breathlessness is the main limiting factor; in endurance events, hyperthermia limits performance, the heart and circulation no longer being able to sustain their task by transferring heat to the skin, providing fluid for evaporation and blood for the brain and active muscles. It is rare that the muscles themselves fail. The maximum work output of a trained cyclist bicycling with one leg is nearly as great as the output when he uses both (Duner, 1959).[36] It appears, therefore, that the oxygen transfer mechanism is the limiting factor; in which case either the heart or lungs must be responsible. The arterial blood oxygen tension at the breaking point of maximal exercise is similar to that at rest (Table I), a sure sign that the lungs are not to blame. On the other hand, the inadequacy of muscle blood supply is shown by the rise of blood lactic acid which accompanies strenuous exercise; this, as A. V. Hill[37] and his collaborators have demonstrated, is a measure of the oxygen debt which is incurred by the muscles. Yet the 'one-leg' experiment suggests that it is not a limitation of the muscle capillary bed which is mainly responsible. Inadequate cardiac output is a more probable explanation. An analagous situation exists in patients with restricted cardiac output, who incur an oxygen debt at a relatively low level of energy expenditure so that when they exercise, their blood lactic acid concentration rises prematurely and they are obliged to stop. In athletes the mechanism is less obvious because a man who is exercising his legs maximally on a bicycle may be able to increase his energy expenditure by using his arms as well (e.g. Nielsen and Hansen 1937).[38] Thus it is likely that there is a balance between a general and local circulatory inadequacy, and this balance is tipped, sometime

TABLE I.

Effect of heavy exercise upon arterial blood oxygen tension in normal subjects

** = significant reduction*

		TENSION (mmHg)			
		REST	EXERCISE		
	Number		Mod.	Heavy	Max.
Asmussen and Nielsen (1958)	11 Students	87	89	82*	87
Mitchell Sproule and Chapman (1958)	24 normal men ages 20–43	85	86	89	91
Holmgren and Linderholm (1958)	14 young racing cyclists	101	—	85*	79*

one way sometimes another, by other factors, such as the duration and intensity of training or the additional strain imposed by a high ambient temperature. In either event, the exercise may be said to be 'circulation-limited'. This is not, however, the whole story. Lactic acid liberated as a consequence of circulatory inadequacy increases blood acidity and is a potent respiratory stimulus. The rise in body temperature due to insufficient skin blood flow has the same effect. The rate and depth of breathing increases and the respiratory minute volume rises. This increased respiratory activity is not matched by a corresponding rise in oxygen intake, since the blood flow through the lungs is already approaching its limit and does not increase proportionally. Instead the ventilation per unit of oxygen consumption rises. Fig. 3 shows this in a non-athletic subject. The excess ventilation serves to eliminate carbon dioxide, and so to correct the change in blood pH produced by lactic acid. It facilitates body cooling by increasing evaporation from the surface of the respiratory tract; but this effect is small. It also augments the venous return to the heart. In these respects increased activity of the lungs minimises the consequences of inadequate cardiovascular function, but at a cost which rapidly becomes

insupportable, partly because the increased energy expenditure required to move the lungs is out of proportion to the increase in ventilation, and partly because great strain is imposed on the respiratory apparatus. This is shown by a decrease in intrathoracic pressure during inspiration. The increased oxygen cost of breathing soon outruns the blood flow available to sustain it, so that the rise in ventilation only serves to increase the lactic acid production and hence the stimulus to ventilation. A second vicious spiral is thus established. The breaking point varies from one individual to another but the final limiting factor is almost certainly respiratory discomfort. This has been investigated by Marshall Stone and Christie (1954)[39] who measured the intrathoracic pressure at the breaking point of maximal exercise in a variety of conditions. They studied normal subjects, patients with chronic lung disease, in whom ventilation on exercise was limited by lung damage, and patients with heart disease who had both a restricted cardiac output and stiff lungs. In these very different subjects the maximum oxygen intake varied between 0·4 and 4·3 l/min. and the ventilation at the breaking point from 20 to 130 l/min. The least variable feature was a drop in intrathoracic pressure on inspiration to minus 20–45 cm. H_2O. The respiratory distress produced by this strain upon the respiratory apparatus is likely to be an important factor determining when the breaking point is reached.

b. *The Effects of Oxygen Breathing*

Oxygen breathing increases maximal exercise ability. This is due both to an increased flow to the muscles of oxygen which is in solution in the blood and to a reduction in exercise ventilation. The additional oxygen only amounts to about 1·7 ml. per 100 ml. of blood, an increase of 8 per cent, but sufficient to reduce the anaerobic metabolism and to lower the blood lactic acid level during strenuous exercise. Oxygen breathing also increases the efficiency of cardiac contraction and reduces the pulse frequency, so that the cardiac output tends to rise. Both these effects, by reducing acidaemia, decrease ventilation on exercise. Oxygen breathing also causes an immediate reduction in ventilation within a few seconds, before there has been any change in the level of circulating lactic acid. This reduction is believed to be a direct consequence of the rise in blood oxygen tension (Dejours, 1959);[40] it is an additional factor increasing exercise performance.

Theoretical consideration of these changes suggests that 100 per cent oxygen is always more effective than lower concentrations in increasing maximal exercise ability. But, Bannister and Cunningham (1954)[41] found on themselves that 56 per cent oxygen is equally effective; and this is true also of patients with chronic lung diseases (Cotes and Gilson, 1956)[42]. The reasons

are not yet understood. It may be that the fall in blood flow to the brain, which is one consequence of 100 per cent oxygen breathing, exerts an adverse effect and that, in these situations, the immediate effect of oxygen in reducing ventilation (for which only a 30–50 per cent concentration is required) is of greater consequence than the indirect effect of the dissolved oxygen upon lactic acid production.

The effect of oxygen illuminates the mechanisms which normally limit exercise. Its use increases maximum exercise ability and hence the maximum oxygen intake. At the same time the ventilation minute volume during maximal exercise is not greater on oxygen than it is on air (Nilesen and Hansen 1937);[38] which again suggests that sensations associated with breathlessness may be limiting activity.

i. *Oxygen During Exercise.* Oxygen has been used to increase exercise ability at high altitudes, where the ambient oxygen pressure is low, and in patients with lung disease, in whom derangement of lung function is associated with a reduced arterial blood oxygen tension. In both situations there is a risk of carbon dioxide accumulation, but this has not yet proved to be a serious deterrent. Oxygen may also be used to improve athletic performance, though it is questionable whether this change is desirable. The equipment is available and oxygen has been breathed successfully during near maximal exercise in many physiological laboratories; but there is a carbon dioxide risk if the wrong procedure is adopted. Hill and Flack (1910)[43] found that if they flushed out the lungs with deep breaths of oxygen then took maximal exercise, holding the breath, they became unconscious. When this occurs it is due to a rise in the carbon dioxide tension in the blood, which in turn is a direct result of the failure to breath. It does not happen when the lungs are initially full of air, because the fall in oxygen tension during breath-holding produces an overwhelming urge to breathe. If the subject wishes to use oxygen he should, therefore, be reminded to breathe, especially if the period of exercise is so short that he may be tempted to abstain. With this precaution, no harm is to be anticipated; and there seems good reason to expect that times for some events could be considerably reduced.

(ii) *Oxygen Before and After Exercise.* The body has only a very limited capacity to store oxygen and the amount which can be accumulated is negligible in comparison with requirements during exercise. Thus a few breaths of oxygen before an event do not confer any physiological advantage except when the subject then uses his lungs as an oxygen reservoir by breath-holding. This is not permissible, as Hill and Flack discovered.

Oxygen breathing after an event might be expected to speed recovery, as assessed by the rate at which the oxygen debt is repaid and the blood lactic

acid level falls. But the rate of lactic acid removal from the blood of patients with moderately severe valvular disease of the heart has been found to be normal and unaffected by oxygen breathing; and this is also likely to be true of athletes. Oxygen is not known to effect recuperation in other ways, but at the time of writing this does not appear to have been specifically investigated. It should be possible to obtain direct experimental evidence by first assessing performances breathing air during a second period of exercise a few minutes after major exertion, then by repeating the experiment breathing randomly allocated mixtures of air and oxygen from superficially identical cylinders. Meanwhile, in the absence of information to the contrary, it seems probable that any benefit conferred by oxygen during recovery from exercise is a result of suggestion. If this is so, it could soon lead to a psychological dependence on oxygen which might be difficult to break. The use of oxygen during recovery should therefore be discouraged until more information becomes available.

c. *The Effects of Age*

Our knowledge of the physiological consequences of ageing has advanced considerably over the last twenty years, the most notable contribution being made by Dr Nathan Shock and his colleagues in Baltimore. Its effects are due to the ravages of time and diet and to the buffeting to which we are all to some extent exposed; but loss of condition due to decreased physical activity also contributes. This is an aspect which readily lends itself to experiment and to studies in human biology. There is still much to learn. In general maximal exercise ability decreases progressively with age (Fig. 11), partly because of a general reduction in the number of active body cells in the muscles and elsewhere; there is, for example, a reduction in the basal metabolism (Fig. 11) and in the volume of the intra-cellular (as distinct from extra-cellular) water in the body. In the lungs there is less elastic supporting tissue available to hold open the small air passages; as a result of this diminution in elastic recoil, the calibre of the airways at a given lung volume decreases and the airway resistance rises; so that the maximum breathing capacity is reduced. On expiration the airways close completely at a progressively increasing lung volume and the volume of residual gas in the lung rises. At the same time the fine balance between pulmonary ventilation and perfusion is disturbed, so that more air is needed for a given uptake of oxygen (i.e. the ventilation equivalent increases) and the pulmonary diffusing capacity diminishes, due to loss of alveolar capillaries. These changes, which are illustrated quantitatively in Fig. 11, affect all aspects of lung function and largely explain the reduction in exercise capacity with age. But they do not occur in isolation. Similar

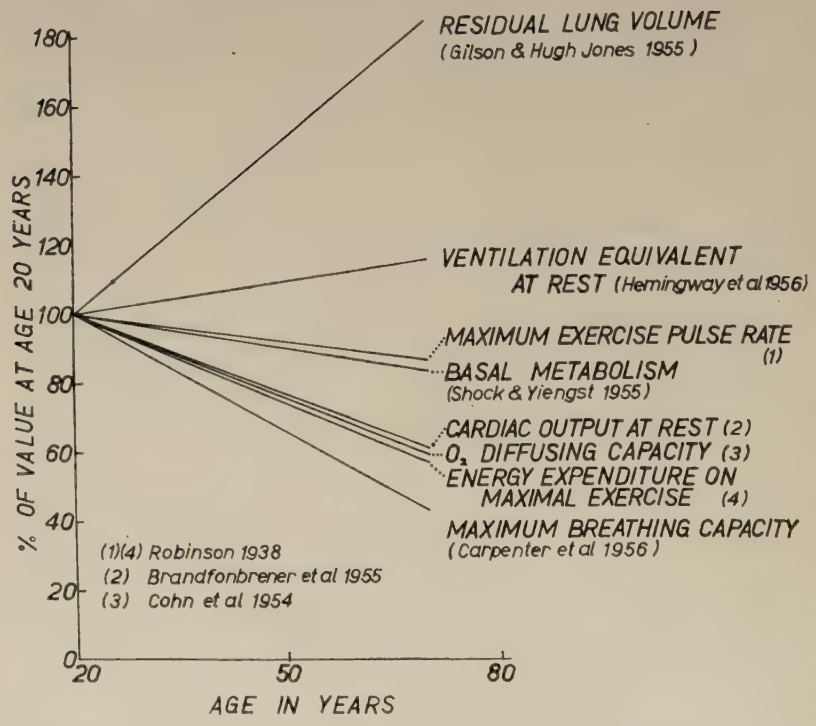

FIG. 11. Percentage change with age in physiological indices for normal men.

changes affect every tissue of the body. The heart's output decreases, both at rest by a reduction of stroke volume, and on exercise by reduction of the maximal obtainable pulse frequency. The smaller cardiac output lessens heat tolerance, and lactic acid appears in the blood at a lower level of exercise. The ability to accumulate an oxygen debt also decreases with age, so that the highest attainable blood lactic acid concentration falls. In the central nervous system the time taken to react to a stimulus increases, and there is a reduction in the rate of transmission of impulses up the individual nerve fibres. Co-ordination is impaired, and this in turn may affect exercise performance. The relative magnitude of some of these changes is illustrated in Fig. 11, which shows that the maximum exercise ability of subjects in good health decreases by nearly a half between the ages of twenty and seventy; this is also true of the function of the heart, lungs and kidneys. For simplicity, deterioration is represented as starting from the age of twenty, which is probably true

of the function of the lungs and kidneys, but less so of organs more amenable to training; there are also big individual variations to take into account. The effect of training is most marked in events demanding a high degree of endurance, in which performance improves well into the thirties, provided that a vigorous training programme is maintained. But the process cannot be maintained indefinitely. Inevitably the effects of age assert themselves and performance eventually falls off. Physical training can do little to slow down the loss of cells which occurs with ageing, but it can do much to slow down its effect upon performance. In this respect physical exercise is likely to stand a man in good stead all his days.

REFERENCES

1. Muller, E. A. 'Training muscle strength'. *Ergonomics* 2, 216, 1959.
2. Crowden, G. P. 'Physiological cost of the muscular movements involved in barrow work'. M.R.C. Industrial Fatigue Res. Board No. 50, *London H.M.S.O.* 1928.
3. Gemmil, C., Booth W. and Pocock, P. 'Muscular training I. The physiological effect of daily repetition on the same amount of light muscular work'. *Amer. J. Physiol.* 92, 253, 1930.
4. Knehr, C. A., Dill, D. B. and Nuefeld, W. 'Training and its effects on man at rest and work'. *Amer. J. Physiol.* 136, 148, 1942.
5. Cotes, J. E. and Meade, F. 'Physical training in relation to the energy expenditure of walking and to factors controlling respiration during exercise'. *Ergonomics* 2, 195, 1959.
6. Kruse, R. D. and Mathews, D. K. 'Bilateral effects of unilateral exercise'. *Arch. Physiol. Med. and Rehabilitation.* 39, 371, 1958.
7. Cureton, T. K. 'Relationship of physical fitness to athletic performance and sports'. *Amer. J. med. Ass.* 162, 1139, 1956.
8. Fletcher, J. G. Personal communication.
9. Taylor, H. L., Henschel, A., Brozek, J. and Keys, A. 'The effects of bed rest on cardiovascular function and work performance'. *J. appl. Physiol.* 2, 223, 1949.
10. Fenn, W. O. 'Work against gravity and work due to velocity changes in running; movement of the center of gravity within the body and foot pressure on the ground'. *Amer. J. Physiol.* 93, 433, 1930.
11. Cotes, J. E. and Meade, F. 'The energy expenditure and mechancial energy demand on walking'. *Ergonomics* 3, 97, 1960.
12. Comroe, J. H. Jr., Forster, R. E., Du Bois, A. B., Briscoe, W. A. and Carlsen, E. 'The Lung'. Chicago, The Year Book Publishers, 1955.

13. Pugh, L. G. C. 'Muscular exercise on Mount Everest'. *J. Physiol.* (*Lond.*) 141, 233, 1958.
14. Bannister, R. G., Cotes, J. E., Jones. R. S. and Meade, F. 'Pulmonary diffusing capacity on exercise in athletes and non athletic subjects'. *J. Physiol* (*Lond.*) 152, 66, 1960.
15. Shepard, R. H. 'Effect of pulmonary diffusing capacity and exercise tolerance'. *J. appl. Physiol.* 12, 487, 1958.
16. Asmussen, E. and Nielsen, M. 'Cardiac output during muscular work and its regulation'. *Physiol. Rev.* 35, 778, 1955.
17. Astrand, P. O. 'Human physical fitness with special reference to sex and age'. *Physiol. Rev.* 36, 307, 1956.
18. Rushmer, R. F. and Smith, O. A. Jr., 'Cardiac Control'. *Physiol. Rev.* 39, 41, 1959.
19. Karpovich, P. V. 'Physiology of muscular activity'. Saunders, Philadelphia, 1959.
20. Mellerowicz, H. Quoted by M. J. Karvonen 'Work and the Heart'. *Medizin Arch. F. Kreislauf-forsch.* 24, 70. 1956.
21. Rook, A. 'An investigation into the longevity of Cambridge sportsmen'. *Brit. med. J.* 1, 773, 1954.
22. Karvonen, M. J. 'Problems of training of the cardiovascular system'. *Ergonomics*, 2, 207, 1959.
23. Monotoye, H. J., van Huss, W. D., Olsen, H., Hudee, A. and Mahoney, E. 'A study of the longevity and morbidity of college athletes'. *J. Amer. med. Ass.* 162, 1132, 1956.
24. Brouha, L., Heath, C. W. and Graybiel, A. 'Step test. Simple method of measuring physical fitness for hard muscular work in adult man'. *Rev. Canad. de Biol.* 2, 86, 1943.
25. Fletcher, J. G. 'Maximal work production in man'. *J. appl. Physiol.* 15, 764, 1960.
26. Ryhming, I. 'A modified Harvard step test for the evaluation of physical fitness'. *Arbeitsphysiol.* 15, 235. 1954.
27. Taylor, C. 'Some properties of maximal and submaximal exercise with reference to physiological variation and the measurement of exercise tolerance'. *Amer. J. Physiol.* 142, 200, 1944.
28. Wahlung, H. 'Determination of physical working capacity'. *Acta. med. scandinav.* 132 (suppl. 215), 1948.
29. Wyndham, C. H., Strydon, N. B., Maritz, J. S., Morrison, J. F., Peter, J. and Potgeiter, Z. U. 'Maximum intake and maximum heart rate during strenous work'. *J. appl. Physiol.* 14, 927, 1959.

30. Astrand, I., Astrand, P. O. and Rodahl, K. 'Maximal heart rate during work in older man'. *J. appl. Physiol.* 14, 562, 1959.
31. Robinson, S. 'Experimental studies in physical fitness in relation to age'. *Arbeitsphysiol.* 10, 251, 1938.
32. Fox, R. H. 'Heart Stress and Athletics'. *Ergonomics* 3, 307, 1060.
33. Bannister, R. G. and Cotes, J. E. 'The effect of changes in enviromental temperature upon body temperature and performance during strenuous exercise'. *J. Physiol.* (*Lond.*) 147, 60, 1959.
34. Newburgh, L. H. 'Physiology of heat regulation and the science of clothing'. Philadelphia, Saunders, 1949.
35. Pugh, L. G. C. and Edholm, O. G. 'The physiology of channel swimmers'. *Lancet* (2), 761, 1955.
36. Duner, H. 'Oxygen uptake and working capacity in man during work on the bicycle ergometer with one and both legs'. *Acta. Physiol. Scand.* 46, 55, 1959.
37. Hill, A. V., Long, C. N. H. and Lupton, H. 'Muscular Exercise, lectic acid and the supply and utilization of oxygen, parts I-III, IV-VI, VII-VIII'. *Proc. Roy. Soc. London s.B.* 96:438; 96:455; 97:84; 97:155; 1924-1925.
38. Nielsen, M. and Hansen, O., 'Maximale Korpealiche Arbeit Bei Atmung, O_2-Reicher Luft'. *Skand. Arch. Physiol.* 76, 37, 1937.
39. Marshall, R., Stone, R. W. and Christie, R. V. 'The relationship of dyspnoea to respiratory effort in normal subjects, mitral stenosis and emphysema'. *Clin. Sci.* 13, 625, 1954.
40. Dejours, P. 'La regulation de las ventilation au cours de l'exercise musulaire chez l'homme'. *J. Physiol.* (Paris) 51, 163, 1959.
41. Bannister, R. G., Cunningham, D. J. and Douglas, C. G. 'The carbon dioxide stimulus to breathing in severe exercise'. *J. Physiol.* (*Lond.*) 125, 90, 1954.
42. Cotes, J. E. and Gilson J. C. 'Effect of oxygen on exercise ability in chronic respiratory insufficiency. Use of portable apparatus'. *Lancet* 1, 872, 1956.
43. Hill, L. and Flack, M. 'The influence of oxygen on athletes'. *J. Physiol.* (*Lond.*) 28, 39, 1909.

3

POSTURE AND THE MECHANICS OF MOVEMENT

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C.V.O., M.B.E., T.D., F.R.C.S.

MOST athletes are aware that there is such a thing as good and bad posture; few, however, realise the importance and significance of good posture to athletic prowess or how much poor posture contributes to indifferent performance and even injuries.

The Oxford Dictionary[1] describes posture as 'The position and carriage of the limbs and the body as a whole'; but posture is as much an attitude of the mind as of the body. A man's spirit shows in the way he holds himself: in depression he slumps, in elation he walks with a spring. 'Correct upright posture' may be described as an attitude of the mind towards the body promoting both mental and physical equilibrium and poise (Tucker, 1960).[2]

The different postural phases may be studied with the body in—

(a) the upright position at rest.
(b) the upright position in preparation for movement.
(c) the upright position in motion.
(d) the sitting or lying position at rest.
(e) the sitting or lying position in preparation for movement.
(f) the sitting or lying posture when one or more sections of the body are in motion.

It is convenient to visualise the human body as a series of mobile parts attached by muscles, ligaments, and fasciae at several relatively firm levels. The body may be compared with a multi-storeyed house, the construction of which is based upon the foundation and ground floor from which it extends upwards. The stability of the house depends on firm foundations and the subsequent proper construction of each storey. So, also, the upright human body depends for its stability upon a number of articulations, the most important of which I consider, for simplicity, at five fixing levels:—

(1) *The Ankle Joint Level:* The whole body is firmly fixed on the ankle joints and feet so that each foot forms the half of a pedestal or base.

(2) *The Hip Joint Levels:* The upper extremities with the head, trunk and

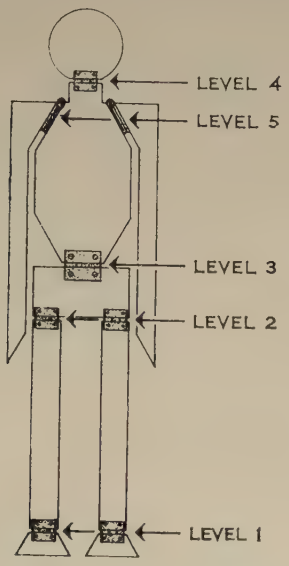

FIG. 1. The five main prime fixing levels of the body.

pelvis move either on one hip joint or along a horizontal line passing through them both. Movements such as forward bending at the hips take place at this level.

(3) *The Lumbo-Sacral Level:* The upper extremities, with the head and trunk, must be firmly fixed at this level, which is where waist line movements take place.

(4) *The Head/Neck Level:* Nodding movement takes place between the occiput and the atlas, whereas in ordinary flexion and extension of the head on the neck most of the movement occurs at the $C_4/5$ or $C_5/6$ levels.

(5) *The Shoulder Girdle Level:* The arms and shoulder girdles, including the clavicle, are firmly attached to the sternum in front and by muscles to the chest wall at the side.

The pull of gravity draws unsupported objects towards the ground. When standing, a man adopts either a symmetrical or an asymmetrical position (Fig. 2). In symmetrical standing the line of gravity falls between the two feet, each foot bearing half the weight of the body; in asymmetrical standing most of the weight is carried on one foot, the other foot simply helping to maintain balance. If the weight is carried forward on to the toes only the calf, hamstrings and back muscles are in active contraction, but if the weight is on the heels, so that one tends to fall backwards, the anterior tibial group, the

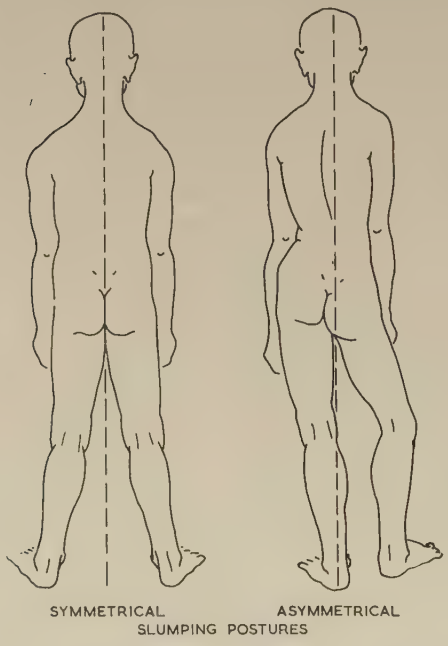

FIG. 2. Symmetrical and asymmetrical standing.

quadriceps, and the anterior abdominal muscles contract slightly and maintain equilibrium.

It is possible to measure the electrical activity of individual muscles or muscle groups (electromyography). In what is usually regarded as the normal upright or stand-at-ease posture, there is minimal activity in the groups of muscles which surround the fixing levels, but a good deal of tension in the joint capsule and overlaying fascia. This tension is the result of the weight of the mobile parts of the body, which, if not held firmly to the fixed parts by balanced muscular activity, pull on the joints, and the effects of strain are felt first in the muscles and later in the components of the joints themselves.

It should be evident, therefore, that the 'normal' stand at ease posture is detrimental to health, as the effects of gravity are constantly working on the body. I call it 'Inactive Slumping Posture' (Fig. 3). It is common in adolescents and, if not corrected, may constitute a bad habit which, in later life, is liable to cause physical suffering.

Also, if an athlete is adopting Inactive Slumping Posture and it becomes necessary to make a sudden quick movement, certain muscles, such as the

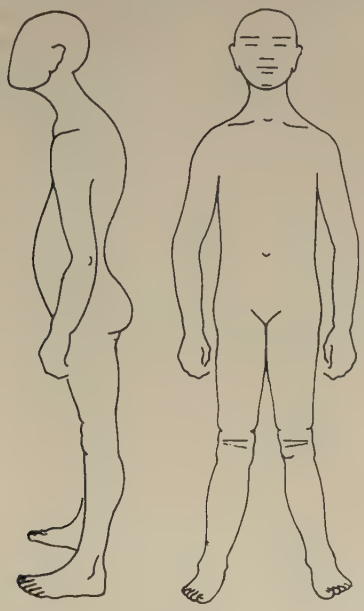

FIG. 3. Inactive slumping: posture in two views.

calf or hamstring muscles, are liable to suffer strain. They are subjected to what is called 'Shock Strain' i.e. the muscular or tendinous fibres are not in a state of preparedness to accept the sudden strain of the body weight and, therefore, there may be partial or complete rupture of the muscle or tendon.

In direct contrast to this negative sort of posture is what I call 'Active Alerted Posture', (Fig. 4) in which electromyographic studies show there is balanced action between the muscle groups at each of the fixing levels, so that the weight of the mobile parts is carried by these muscles. Thus, there is no chronic strain upon the ligaments and the other components of the joints. These, therefore, are far less likely to become deranged in middle or old age.

There is a further postural attitude, which I call 'Passive Supported Posture'. When the body is at rest, either sitting or lying, each mobile part is supported in the neutral position. If one sits in a chair which has arm rests, each section of the body is supported in a neutral position, so that the chair takes the weight of the mobile parts, without either a stretching or a compressing force upon any joint (Fig. 5). The one exception is the neck; without a

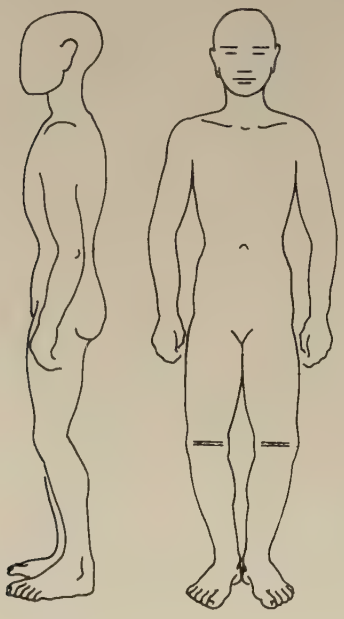

FIG. 4. Active alerted posture in two views.

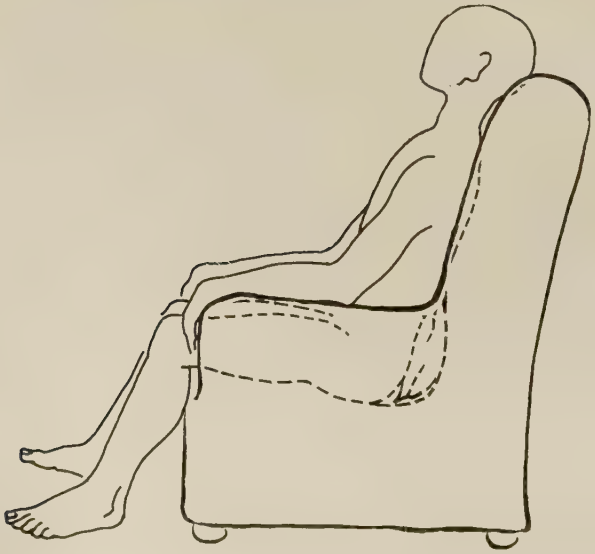

FIG. 5. Passive supported posture, sitting.

head rest the head must be held actively supported by the neck muscles, or there will be a strain upon the joints—a strain familiar to anyone who has fallen asleep in a chair. In bed, where the body is similarly supported in the 'Passive Supported Posture' it is important to prevent strain on the cervical joints by positioning the head on several pillows so that it is held in a neutral position. Four pillows, arranged in an inverted 'A' best serve this purpose; whether one sleeps on the back or the side, the head is held in a neutral position (Fig. 6). In 'Passive Supported Posture' in bed there should be no activity necessary in any muscle: but before rising or making any purposeful movement, one should alert the joints and muscles by adopting 'Active Alerted Posture' in the part to be moved, in order to avoid strain upon the joints. This is normally an automatic preparation for movement; but if there

FIG. 6. Passive supported posture, lying with the joints in a neutral position.

is inflammation in any joint, one should make a conscious effort to alert the muscles or the movement may cause pain.

The division of posture into 'Active Alerted', 'Inactive Slumping' and 'Passive Supported' may be criticised as artificial, but these attitudes cover all the positions of the body whether upright, sitting or lying, at rest or in motion.

Therefore, the difference between Active Alerted and Inactive Slumping Posture in the upright positions is simply that in the former all the muscles at the fixing levels are in a state of balanced activity, and the effects of gravity are lessened; in the latter the weight of the mobile parts of the body is unsupported, with consequent strain upon the muscles and joint structures.

It may be objected that a man will wear himself out by the continual muscular effort of 'Active Alerted Posture'. Electromyographic tests during this posture show electrical activity in all groups of muscles. When the posture is first adopted the intensity of electrical activity rises; but as the amount of activity in each group of muscles is the same, the joints are in balance without any fatigue. Therefore, those who practise Active Alerted Posture at all times

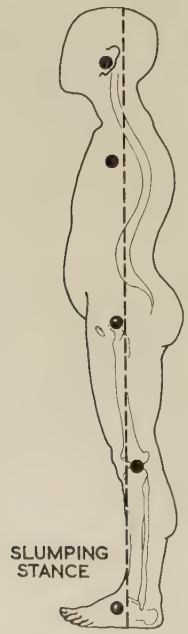

FIG. 7. Line of gravity in inactive slumping posture.

(except, of course, when they are in the Passive Supported Posture) find that they tire far less. If they allow themselves to adopt Inactive Slumping Posture, they immediately feel the dead weight of the mobile parts with a consequent sensation of strain.

Inactive Slumping Posture

In Inactive Slumping Posture, a man stands with his weight on the inner side of the foot, mainly on the heel and the metatarsal heads. His knees are hyperextended; his abdomen protrudes, there is an accentuated lumbar hollow, with the buttocks prominent. The hips are also in an extended position, and the head falls forward with the chin protruding and tilted upwards. If the gravity line of each segment of the body were joined it would be a crooked line (Fig. 7). Since the effects of gravity are not counteracted, there is strain, first on the muscles at the various fixing levels, then on the joints. There are secondary effects on the blood vessels, and on the abdominal and thoracic contents.

The Harmful Effects of Inactive Slumping Posture

A. At the ankle joint level Inactive Slumping Posture can cause, directly or indirectly:—

1. muscle strains, which manifest themselves as strains, either of the longitudinal or anterior transverse arch, or both.
2. in older people, symptoms caused by arthritis of the ankle, mid-tarsal and metatarso-phalangeal joints.
3. stress fractures of the metatarsal bones, fibula and tibia.
4. vascular disturbances, such as varicose veins.
5. secondary strains on the knee, hip, or low back.

B. At the hip joint level muscular, and subsequently joint, strains lead to osteoarthritis.

Trueta[3] and Denham[4] have shown that in the 'stand easy' position, the hip is held extended and, because of tension of the Y-shaped ligament and the over-lying muscles, the head of the femur is held like a nut in a nut-cracker. Excessive wear and tear of the femoral head is inevitable.

C. At the lumbo-sacral level there may be low back pain of either muscular or articular origin at either L4/5 or the L5/S1 levels. The fulcrum for forward bending of the trunk is here, and the intervertebral discs, being the most vulnerable components of the spinal segments, are particularly liable to injury. They also suffer most in lumbo-sacral spondylosis. The pendulous abdomen may also be a potent cause of lumbo-sacral strain.

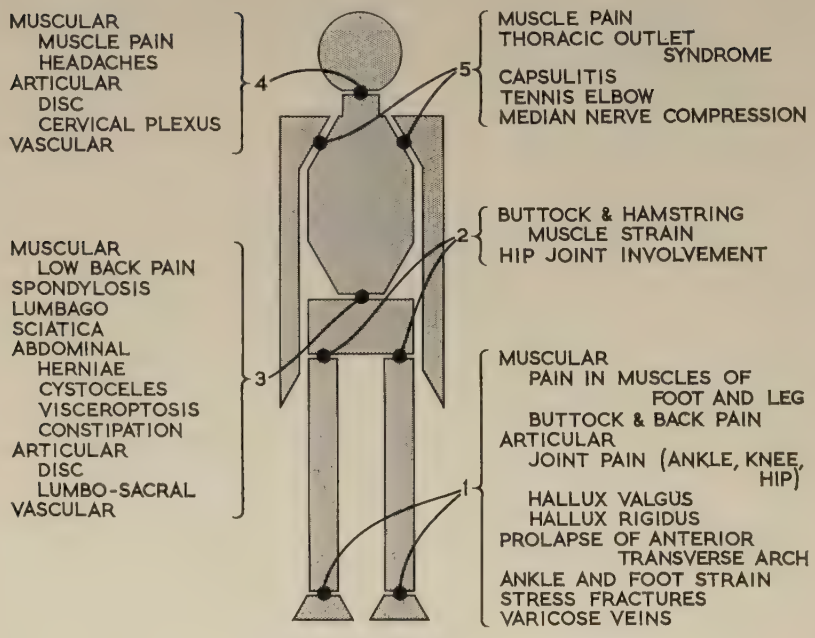

FIG. 8. The effects of postural strain at the five main fixing levels.

In front, the sagging of the abdomen strains the anterior abdominal muscles so that herniae or cystoceles often develop. There may be visceroptosis, with obstinate constipation.

Secondary vascular disturbances, even of the cord itself, are sometimes associated with degenerative changes of the discs at the L4/5 and L5/S1 levels.

D. At the head/neck level there may be symptoms of muscle and joint strains. These involve the nerves of the cervical plexus and present as aches and pains at the base of the skull and neck. Vascular disturbances through secondary involvement of the vertebral artery may account for vertigo, peculiar cerebral disturbances, and even fainting.

E. At the shoulder girdle level there may be the signs and symptoms of a thoracic outlet syndrome, which may be associated with capsulitis of the shoulder, forms of tennis elbow (epicondylitis), acroparaesthesiae and even medial nerve compression in the carpal tunnel. Although there may be anatomical and pathological causes for the syndrome, such as cervical rib, anomalies of the scalenus anticus and medius, abnormal fascial thickenings and anomalies in the formation of the posterior cord of the brachial plexus, a

slumping posture will accentuate the thoracic outlet syndrome and produce symptoms at an earlier age.

The constant drag on the shoulder girdle muscles may in time throw strain on the lower cervical vertebral joints and produce symptoms of cervical root compression.

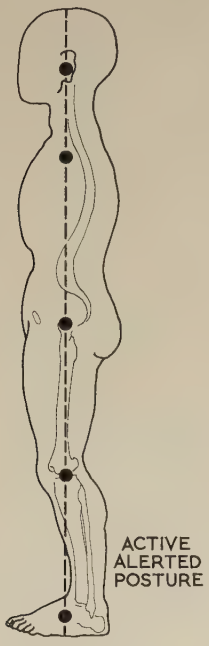

FIG. 9. Line of gravity in active alerted posture.

Active Alerted Posture

The evils that stem from poor carriage can be avoided by Active Alerted Posture. In this the weight of the body is carried on the outer side of the foot and the toes grip the ground. The inner border is braced for action, with balanced action of all the muscle groups of the foot, ankle and calf. The knee joints are held very slightly flexed so that there is balanced action between the quadriceps and hamstrings. The joint is thus protected from gravity strains. Similarly, slight flexion at the hip and the balanced tone of the muscles prevents the nut-cracker effect described above.

The abdominal muscles are held up and in, a position which is automatic

if one thinks of the navel as a 'pressbutton' to be clipped to the vertebral column. The buttocks are firm and the back flattened: as a result, the vertebral column is flattened, being neither hyper-extended as in the extreme 'military' position, nor flexed as in the slumping posture.

The chin is pulled in by slight contraction of the sterno-mastoid and the anterior cervical muscles, and so balance the posterior spinal muscles. The shoulders and shoulder girdles are held up and slightly forward, so that there is no gravity strain either on the joints of the girdle or upon the muscles of the cervical spine. (Fig. 10.)

With a little practice one learns to hold this posture and yet remain entirely at ease. This is, in fact, the position of the athlete who looks quite relaxed but

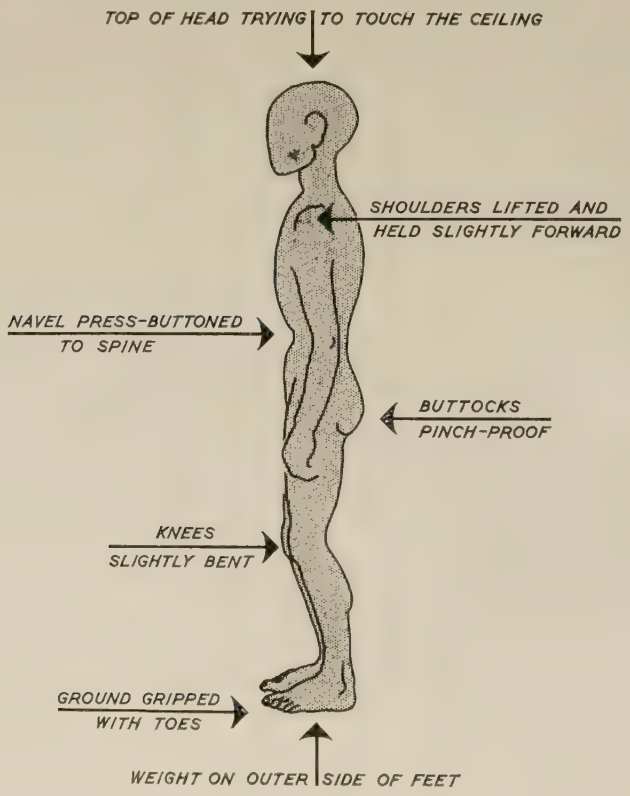
TOP OF HEAD TRYING TO TOUCH THE CEILING
SHOULDERS LIFTED AND HELD SLIGHTLY FORWARD
NAVEL PRESS-BUTTONED TO SPINE
BUTTOCKS PINCH-PROOF
KNEES SLIGHTLY BENT
GROUND GRIPPED WITH TOES
WEIGHT ON OUTER SIDE OF FEET

FIG. 10. This illustrates the various positions of the body in perfect active alerted posture.

also ready to spring instantly into action. At all the prime fixing levels, the muscles are in balanced contraction, which not only resists the effects of gravity, but also protects the joints from the strain of sudden movement.

The Mechanics of Movement

It is important for a doctor who attends athletes to have some knowledge of the mechanics of muscle action or kinesiology.

There are three orders of lever action depending on the position of the fulcrum or point of pivot and the force and load. The first order is easy to understand; it is the principle of the see-saw in which the fulcrum or pivot point is in the centre, the load at one end and the force at the other. The see-saw which has a child at each end illustrates how the terms load and force are interchangeable. When a man bends his back to pick something up, the fulcrum pivot is centred at the L_5/S_1 intervertebral disc. The erector spinae

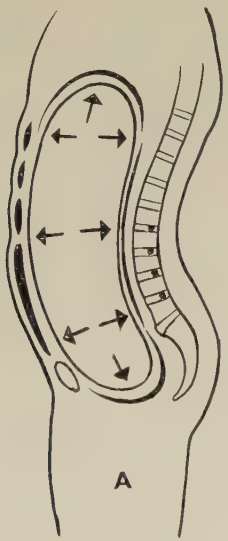

FIG. 11. Upright, Active Alerted.

The intra-abdominal pressure by contraction of the abdominal muscles, diaphragm and pelvic diaphragm muscles, is evenly distributed and prevents strain on the vertebral joints and discs.

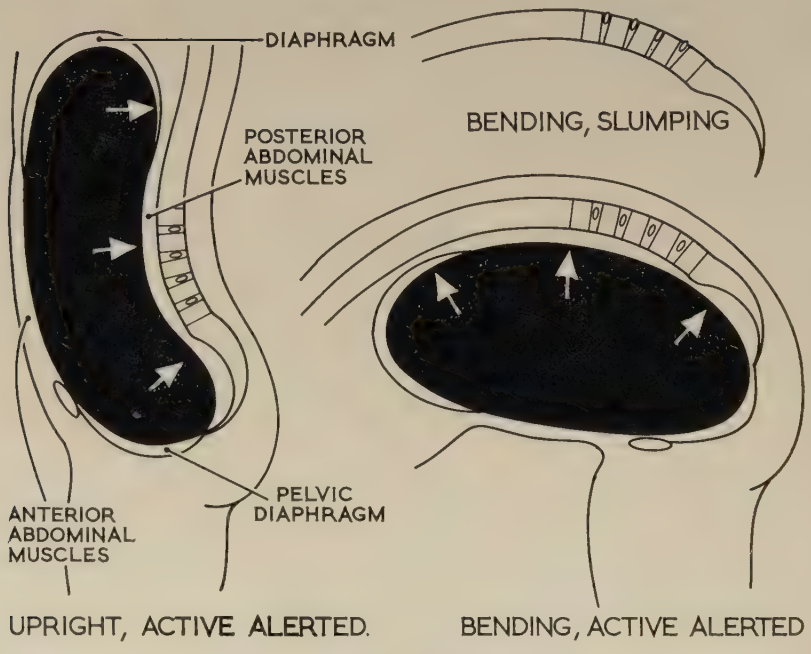

FIG. 12. Bending, Active Alerted.
In bending the action is an active movement instead of an inactive slump, and the trunk and pelvis bend together so that the pivot is through the hip joints. The abdominal pressure, by the action, is maintained in equilibrium and there is no strain on the intervertebral joints and discs.

muscles which lift the object (the load) are working only about two inches from the fulcrum. If the object is well in front of him—say thirty inches—the strain on the pivot point is magnified some fifteen times; and in addition he must lift the weight of his head, trunk and arms. If the object weighs 50 lbs., the force working on the intervertebral disc is about 800 lbs. (Fig. 11.) In Active Alerted Posture the action of bending is a positive one, carried out by contraction of the abdominal muscles. These should be so firmly contracted that, with the diaphragm and muscles on the posterior abdominal wall, they form a wall of firmly contracted muscle, which supports the spinal column and its vulnerable intervertebral discs. (Fig. 12.) The intra-abdominal pressures caused by this contraction is like that in an inflated football.[5] On the other hand, if the bending of the trunk is a passive, gravity assisted movement, without balanced muscular contraction, then there is pressure on the front of the vertebral bodies, and a tendency for the nucleus pulposus of

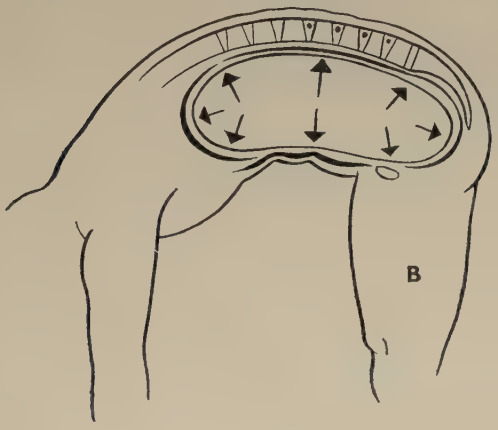

FIG. 13. Bending, slumping.
The force which causes the bend is the weight of the trunk and the pelvis and a strain takes place at the lumbo-sacral level. The weight of the trunk causes pressure on the fronts of the vertebral bodies, tending to displace the nucleus pulposus backwards.

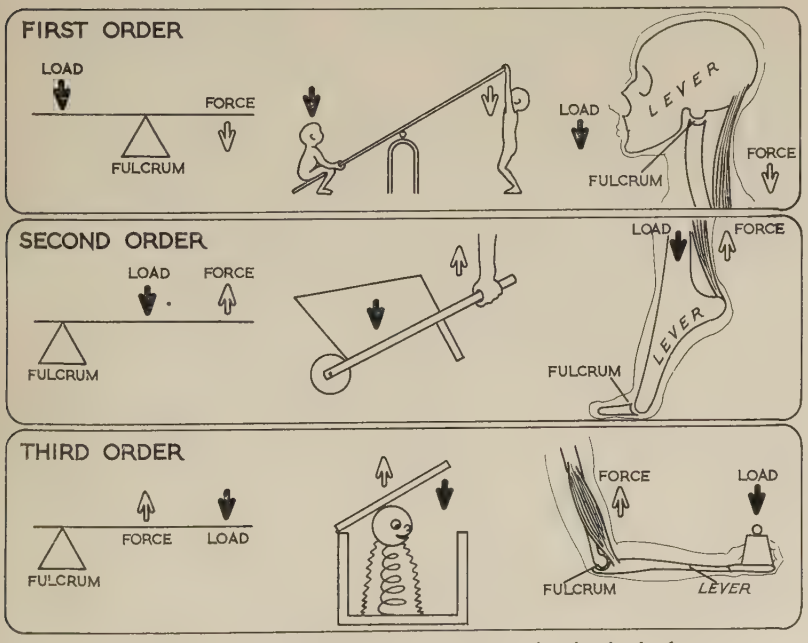

FIG. 14. The different orders of lever action in the body.

the intervertebral disc to be displaced backwards or even to rupture through the posterior longitudinal ligament.

Another example of the first order of lever action is the nodding movement at the occipito-atlantoid joint. (Fig. 14.)

The second order of lever action is illustrated by the wheel-barrow, where the force is at one end, the pivot at (Fig. 14) the other, and the load in the centre. When one stands on tiptoe, the pivot is the toes, the load the body's weight, and the force is exerted by the calf muscles at the other end.

The third order of lever action is the one the body uses most: the load is at one end, the pivot at the other, and the force is in between. When we lift something in the hand, the load is the weight of the article, the lifting force is exerted by the biceps, and the pivot is the elbow joint. (Fig. 14) When we kick a football or hit a ball at tennis or cricket, this is the order of lever action we employ. The ball is the load, the arm or leg of one side supplies the force, which works on a fulcrum supplied by the rest of the body.

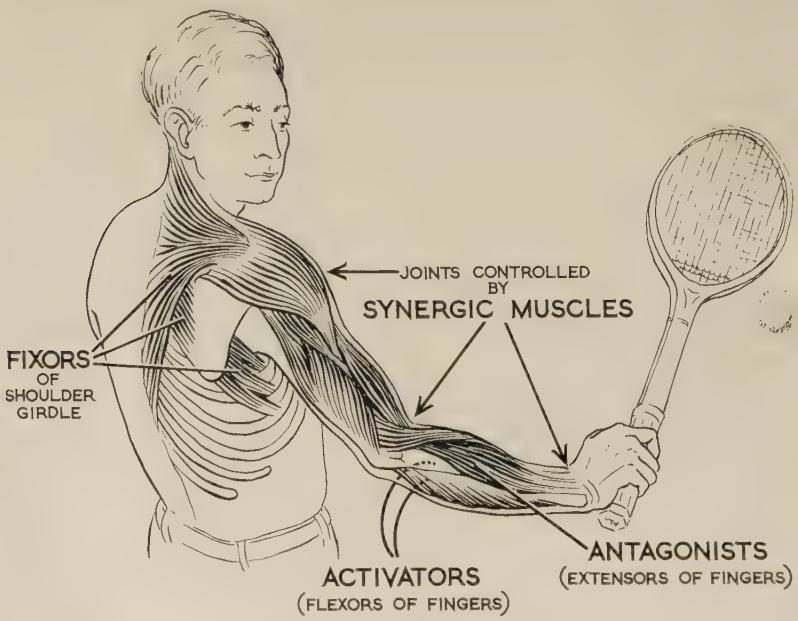

FIG. 15. To demonstrate how the correctly executed backhand tennis shot is performed by the activators acting in conjunction with the synergic and prime fixor groups.

Muscle Action

When a golfer plays a shot, his club and his arms together provide the activating part of the action; the trunk and pelvis act as synergists. The fulcrum is centred at the left hip joint which should be fixed and rigid by contraction of all the muscles of the left buttock, thigh and leg.

The muscle group which initiates an action is called the 'activator group'. The activator group are composed of 'agonists' and 'antagonists'. In a tennis shot, (Fig. 15.) for example, the handle of the racket is gripped firmly by the flexors of the fingers and thumb, which are the agonists. Their opponents, the extensor muscles of the fingers and wrist, are the antagonists. In this type of action the antagonists relax and lengthen (the reciprocal innervation of Sherrington). Sometimes the antagonists become active and lengthen, a process called 'eccentric contraction'. This occurs when the prime mover acting on the joint is the force of gravity; the antagonist muscle contracts and pays out and, as it increases its length, controls the force of gravity—for example, when a man allows the weight of the trunk, head and upper extremities to be the activating force in forward bending. At the beginning of the movement the extensor muscles of the back contract and the erector spinae lengthens, helping to control forward bending. We know that in this action these muscles stop working half way through the action of bending when, therefore, the full body weight is thrown on the lumbo-sacral discs. The antagonists are used also in 'co-contraction'; and this co-contraction of each group of muscles at the prime fixing level occurs in Active Alerted Posture.

The muscles which place the activator groups in the best position to perform an action are called 'synergists', because they work with the activators. When a tennis player hits the ball, his activator muscles are aided by many synergists. The pure anatomist would probably confine the term, in this example, to the wrist fixing muscles: but the shot cannot be played unless the elbow and shoulder are properly positioned, and so it seems reasonable to include the elbow and shoulder muscles as well.

The prime fixor muscles of the shoulder girdle firmly attach the arm, which makes the shot, to the rest of the body. It is, therefore, essential that the feet should be firmly and correctly positioned, so that the trunk, pelvis and legs form a rigid fulcrum on which the arms (through the shoulder girdle) can exert the maximum of effect.

Whatever the game, the distance the ball travels depends on synchronising all the muscle groups, so that the activators are accurately assisted by the synergic groups and perform together on a steady firm prime fixing level. The steadier the prime fixing level the greater the recoil and, therefore, the greater the distance the ball will travel.

Joint Movement

All freely movable (diarthrodial) joints have two main types of movement: voluntary movements, executed by the individual himself: and a range of accessory involuntary movements which are outside voluntary control.[6] The involuntary movements allow for free gliding, twisting, rotations and side to side movements of one bone on another, and only if they have full range can the joints work to their maximum efficiency. The active movements possible at the wrist, for example, are palmar-flexion, dorsi-flexion, and ulnar and radial deviation, but if all these movements are to have their full range the proximal row of the carpal bones must glide forwards and backwards and rotate and pivot from side to side on the lower end of the radius and ulna. In the shoulder joint[7], there are three types of movement—

(1) abduction and adduction of the humerus with its head centred in the glenoid fossa of the scapula.

(2) medial and lateral rotation through the same central axis.

(3) a range of gliding movements so that the head of the humerus slips forwards, backwards, upwards and downwards in the glenoid. When we push a hand into the small of the back we are carrying out a very complicated action which is only possible if all these movements are unrestricted. If a joint has a limited range of movement, the manipulative surgeon is at pains to restore full involuntary movements before all else; because he knows that without this freedom, voluntary movements may tear the joint capsule and ligaments. When the freedom of involuntary movement is established, the full range of voluntary movement can follow careful stretching manoeuvres.

Movement in Sport

So far we have been discussing simple and uncomplicated movement, in order to make clear the fundamental principles. The movements of the athlete, however, are seldom simple. Capener points out that intricate movement is always in a twisting, curving direction. Even in running or walking, flexion and extension of the hip joint are combined with some degree of rotation, adduction and abduction. Moreover, there are similar mechanisms at work in the joints of the feet, knees, spine, and arms. The athlete, pole-vaulting, running or jumping, has no time to think of all the components of a perfect movement: but it is important for his doctor and perhaps his trainer to have a working knowledge of the mechanics of joint movement, so that if there is a breakdown in the mechanism of a particular action, they can put it right.

Let me quote the example of a famous English right hand fast bowler who had experienced recurrent attacks of pain in the under surface of the spring ligament and tarsal scaphoid of the left foot. During the winter, the pain dis-

appeared, but it returned each summer, getting progressively worse during the cricket season. As he was a right-handed bowler he finished his action by planting his left foot forward flat on the ground, twisting the foot towards the left so that the foot and ankle were everted and externally rotated. On examination, he was found to be suffering from a traumatic tenosynovitis of the tibialis posticus, and strain of the spring ligament and the short muscles of the longitudinal arch.

He was told how to correct his ankle and foot posture. As he was taking the final step with the left foot, just before delivering the ball, he learnt to plant the leg firmly on the ground, placing the weight on the outer side of the foot and at the same time gripping with the toes. This had the effect of fixing the ankle and foot firmly to the ground and prevented the foot twisting into a valgus position.

From that time on he had no more pain and he found that his bowling improved. He had learned to distribute his weight on the proper weight-bearing part of the foot and at the same time to fix the ankle and foot firmly. In this way, his right arm and the rest of his body were made to function on a firm prime fixing base.

By going carefully into the events leading up to an injury the doctor can often determine the exact mechanism of the trauma and tell the athlete how to avoid a similar accident in the future. Moreover, when the athlete is ready to return to his sport, the doctor can help him to avoid strain to the injured joint. Most steeplechase jockeys who have fractured a clavicle, for example are able to ride about two weeks afterwards, provided the shoulder movements are reasonably full and the fractured ends are supported by padding and clavicle braces: but, to avoid strain on the clavicle, the jockey is taught to divert the strain from the clavicle and the shoulder girdle generally, by fixing his shoulder and elbow joints firmly. In this instance, the prime fixing level in the upper extremity is transferred from the shoulder girdle down to the elbow and shoulder joints, the shoulder girdle being held rigidly to the rest of the trunk so as to form a part of the body. It can be seen from this that the terms 'activators', 'synergists' and 'prime fixors' are interchangeable, according to the circumstances. When someone does a handstand, the hands and arms are the prime fixing levels and the rest of the body is the activator.

Such nicely co-ordinated movements require a good deal of synchronisation from activators, synergists and prime fixors. They require, also, a full range of both voluntary and involuntary movements at each separate joint. If there is a failure of any one of these component parts, an accident or an injury becomes probable.

Types of Fibres in Striated Muscles

Striated muscles have two functions, to steady the fixing joints and, secondly, to move the mobile parts on the fixed parts. They have two types of fibre—dark red and pale.[8] In man, all striated muscles possess a varying amount of dark and pale fibres, so that each muscle functions either as a prime fixor or as an activator. It is possible to improve the power of a muscle by remedial exercises or by weight lifting, provided the exercises are properly graded. Progressive weight lifting helps to strengthen the static elements, and these days even the sprinter performs modified weight lifting exercises in the course of his training.

Integrated Movement

By combining exercises of both static and dynamic nature with the correct positioning of the various sections of the body—as in Active Alerted Posture—many pathological conditions can be avoided; or, if they occur, can be corrected and recurrence prevented. Yet muscles and joints cannot be considered in isolation. Perfection in movement necessary for modern sport is only possible if all the systems of the body—nervous, cardiovascular, pulmonary and glandular—are fully efficient and in harmony.

Let us complete this discussion of posture with a summary of the various positions the body can adopt, and the best posture for each:

(a) the upright position at rest—Active Alerted Posture.

(b) the upright position before movement—Active Alerted Posture.

(c) the upright position in motion—Active Alerted Posture.

(d) the sitting or lying position at rest—Passive Supported Posture.

(e) the sitting or lying position when one or more sections of the body are able to move—these sections must be alerted into Active Alerted Posture before the movement begins.

(f) sitting or lying posture when one or more sections of the body are in motion—Active Alerted Posture for those sections which are moving.

REFERENCES

1. *Shorter Oxford English Dictionary*. 1955. 3rd Edition. C. T. Onions, Oxford University Press.
2. Tucker, W. E. (1960) *Active Alerted Posture*. Edinburgh: E. and S. Livingstone.
3. Harrison, M. H. M., Schajowicz, F. and Trueta, J. (1953). Osteoarthritis of the Hip: A Study of the Nature and Evolution of the Disease. *J. Bone Jt. Surg.* 35B, 598-626.

4. Denham, R. A. (1959). Hip Mechanics. *J. Bone Jt. Surg.* 41B, 550-57.
5. Bartelink, D. L. (1957). The Role of Abdominal Pressure in Relieving the Pressure on the Lumbar Intervertebral Discs. *J. Bone Jt. Surg.* 39B, 718-725.
6. *Gray's Anatomy*, 32nd Edition, 1958, London.
7. Saha, A. K. (April, 1958). Zero position of the gleno-humeral joint: its recognition and clinical importance. *Annals. R. Coll. of Surgeons*, Vol. 22, No. 4, April 1958, p. 223-236.
8. Buller, A. J., Eccles, J. C. and Eccles, R. M. (1960). Interactions between Motorneurones and Muscles in Respect of the Characteristic Speeds of their Responses. *J. Physiol.* 150, 2, 417-439.

4

FITNESS AND TRAINING

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1. INTRODUCTION

THE NUMBER of terms in general use to describe physical states is confusing. Health, for example, was defined as the 'absence of disease'; but the meaning ascribed to health by the World Health Organisation is more comprehensive: 'a state of physical, mental and social well-being'.

Similarly, fitness means different things to different people. Some regard it as synonymous with health; but, in fact, although the two states have much in common and often exist together, they are not identical. Two people may run for the same bus. The first, a Rugby three-quarter, may arrive before the bus has travelled ten yards, and may show no signs of effort. The other, an accountant of sedentary habits, may need twice the time or distance to catch up; and he may puff and blow as though he had made a major effort. The three-quarter is obviously fitter than the accountant: but he may not be healthier nor even as healthy.

The word 'fit', as in this example, nearly always implies 'fitness for some specific activity'; and therefore carries a suggestion of practice or training. It nevertheless takes health as its starting point; so that the total meaning of a fit man is one who has subjected a healthy body to successful training for a specific activity.

The first step in proficiency at any sport, therefore, is a healthy body. This principle ought to be applied in its very widest sense; whether the defect is anatomical, such as a deflected nasal septum which interferes with breathing, physiological, such as insufficient acclimatisation to new conditions of temperature and humidity, pathological, such as infection of the nasal sinuses, functional, such as inefficient posture, or traumatic such as a damaged limb, until corrected the effort of training cannot be fully effective.

There are three main sorts of training: the general training at the beginning of a season: the training for a special event: and the training necessary to get a man back to full activity after an accident.

2. GENERAL TRAINING

At the beginning of the season, a healthy athlete requires a period of time in which to raise the level of his physical activity to a point which is short of, but in sight of, the ultimate goal—the special event. This process needs four to six weeks.

If he is overweight he must reduce, and this is best accomplished by lowering the caloric intake. He should raise his protein intake at the expense of potatoes, bread, sweets, and other carbohydrate foods. There is no need to reduce fluids, except, of course, that drinks like beer or cider should be taken only in reasonable quantities. The extra exercise of training also helps to reduce weight, but it is unwise to take off more than 3 lbs. a week. We usually consider that for the loss of each pound of body weight 3500 calories of energy have to be expended. An athlete in full training needs between 4000–6000 calories each day; so if he can deny himself 500 calories (for example by avoiding three pieces of bread and butter) he will lose $\frac{1}{7}$ lb. a day, or just 1 lb. each week, the recommended amount.

FIG. 1. The ability to think only of the task in hand is characteristic of all first class athletes. The Cambridge crew of 1961 provides a study in concentration.

(a) *Graduated exercise:* The easiest and most certain way to work up to full activity is in gradual stages. It is best to start off by dividing the training into weekly programmes, the first week being taken up with walks interspersed with short sharp runs, the essential factor being graduated exercise. One may start off, for example, with a three mile walk, interspersing short sprints of fifty yards without maximal effort and with brisk walking bouts. The ratio of sprinting to walking should gradually be increased. If, the next morning one

feels no undue stiffness, the distance can be increased one mile each day, and exercise with a skipping rope added.

Some stiffness is inevitable, but it usually yields to hot baths and hard rubbing with a coarse towel. If it is possible to enlist the aid of a good masseur so much the better. There are people who believe, however, that atheletes are now more prone to injury because they have organised massage. A. V. Hill[1] has suggested that stiffness is due to small, even microscopical tears in the muscles and the areolar tissue. Another cause of stiffness can be the accumulation of metabolites and waste products as the result of muscle action. Whatever the cause, excessive stiffness at the beginning of training is a sign that the level of exercise should be held constant for at least one more day.

By the end of the first week, most athletes have worked up to walking and running at least five miles with periods of fast skipping and bouts of short sprinting.

The second week's training depends on the type of athletic event in pros-

FIG. 2. These athletes are obviously 'keyed up' to use the last ounce of physical energy.

pect. If, for instance, sustained efforts are needed, such as in football or boxing, it may be found best to increase the runs up to ten miles, with bursts over half a mile, in order to promote speed. The amount of skipping can also be increased.

(b) *Postural training:* Throughout Chapter 3 the importance of an active attitude towards posture has been stressed; and as quickness of movement depends on the active supporting action of the prime fixors so that the synergists and activators can come into purposeful action without delay, this attitude to posture must obviously be an important factor in training. It will not only help the athlete to carry out his postural movements correctly and quickly but may also help him to avoid accidents.

c. *Specific exercises:* The second week is also the best time to start specific exercises: the boxer should start using the punch bag and possibly sparring; the footballer should practise running and kicking a ball, starting with a moving ball, dribbling and punting. By the end of this week the athlete should begin to feel a sense of well-being.

In preparation for winter sports the Ski Club of Great Britain has brought out an excellent small book of exercises. This system of training can be of use to athletes in other fields.[2]

Another booklet entitled *Keeping Fit for all Ages* gives excellent advice about fitness in general, covering diet and graduated exercises.[3]

The third week should be taken up with increasing the range of all types of exercises, paying particular attention to the special exercises required; and at the end of the third week a short practice game should be undertaken, or a race run. If the athlete is still grossly overweight one might consider advising a Turkish bath, but weight reduction by dehydration tends to be debilitating reducing reserves which may be needed later in the season.

By the end of the third week, having run a race or played his game, the athlete should feel well and have no undue stiffness. Minor stiffness, the morning after the event, passes off in a day or two.

At the beginning of the season it is better not to play more than two games a week. It is not unusual to see athletes, footballers for instance, who start off the season with great vigour, playing two or three times a week but who, by the end of November, are almost stale.

By the end of the sixth week, the athlete has played in several events and should be about 90 per cent fit. If he has a special event in mind he should try to remain at this level until the time comes to raise himself to the peak of his training at the right moment.

The late Dr Lovelock and, recently Zatopek, Roger Bannister, Chris Chataway, Kuts, Brazier, Landy, Ibbotson, Elliott, and many others have

emphasised how important it is that the athlete should decide well in advance the event for which he wants to be at his peak and he should plan his training programme accordingly.

For this event he wants to be 100 per cent fit, not only physically, but mentally and psychologically as well. An athlete can be physically in perfect trim and yet be mentally below par. Somehow he must try to get his morale high at the right moment. Plenty of sleep is essential. Every athlete requires at least eight hours rest, although this may not be taken up entirely by sleep. Relaxation exercises, as taught to women at antenatal clinics, are also excellent. Not only do they promote physical relaxation but, in some curious way, they bring tranquillity of mind.

It is impossible to lay down rules for the final toughening-up process. It must be left to the individual to devise a programme which suits his own needs. For instance, the boxer who has to take part in a world championship fifteen-round contest will obviously train differently from the 100 yard sprinter. Each, however, must work out a system of training so that he is at his peak on the day of the particular event. It is probably better to be slightly undertrained rather than risk forcing or overtraining and becoming stale. Fresh air, good food and early nights will go a long way to contribute to the highest degree of physical and mental fitness. Moderation in all things is the key to perfect fitness.

Alford,[4] the famous pre-war miler and now chief national coach for Rhodesia and Nyasaland, has stated that the present day athletes train at least five times as hard as those before the War. This may account for the phenomenal improvement in recent athletic results. He describes the basic principles of athletic training centred around three main types of exercises which are designed to improve strength, speed, stamina and skill.

The first : weight lifting. Whereas the shot-putter will train in lifting weights up to 1 cwt. the sprinter works on smaller ones of 20 lbs. It would seem that the action of weight lifting improves the power of the static muscles and thereby the prime fixors. Under the auspices of The Amateur Atheletic Association a small book on Weight Lifting for Athletics has been produced which can be of great use to athletes in other fields of sport.[5]

MacQueen states:—'Highly repetitive movements performed against little resistance demand little cerebration. On the other hand intense concentration and consequently a high degree of cerebration is necessary when trying to overcome heavy resistance. In this way progressive resistance exercise improves neuro-muscular co-ordination and motor-learning.' He considers it an essential that basic weight resisting exercises should form an important part of modern training in every type of athletic event. He describes three funda-

mental methods of training with weights:—(1) training for increased muscle power, (2) training for increased muscle size, and (3) a mixed system in which the exercise principles of these first two methods are used together in varying proportion.[6]

The second: circuit training. This implies carrying out a series of exercises in a room designed to contain about twelve different types of exercise equipment. The athlete will do each one a certain number of times and pass on to the next, until he has performed a dozen different exercises a prescribed number of times in a certain period. His time is gradually reduced as he improves.

The third: the Norwegian 'Fartlek', (Interval Training). This is on the principle of boy scout running and walking exercises. The athlete undertaking a four minute mile accomplishes a fast quarter and then gradually increases the time of the slow quarters until he accomplishes a mile in four minutes.

Bleasdale[7] has applied this to swimming as a Controlled Interval Method of Training. He describes the swimmers' year as composed of a resting phase or stocktaking between October and January, when the competitions have ended; a pre-competition phase from January to March; and a competition phase from April to September. During the first phase the athlete should have a medical and dental check-up and any vaccinations needed; review his stroke technique, and do general training including circuit training, weight training and play games if he wishes. During the second phase, less weight and circuit training are undertaken, speed is increased on shorter distances, and turning and starting are practised. During the third competitive phase the controlled interval method of training is used, which consists of repeated speed work on short distances, and the pace and intensity of work is increased. By these methods the swimmer learns to overcome fatigue.

3. TRAINING FOR THE SPECIAL EVENT

Franz Stampfl[8] in a series of three articles in the *Observer* stated that the whole art of training is learning exactly what is enough. The athlete must get to know himself. He must practise self-discipline in everything that he does. In this way he can condition himself in training to do more and more. Eventually when the great day arrives for a super effort he can perform beyond his previous best.

He advocates training and more training so that the body by repeating and graduating the intensity of effort adapts itself to increasing stress; just as the miler is encouraged to increase speed by running half and quarter miles, so too the sprinter may benefit by including longer distances up to half a mile in his programme of training.

The British Medical Journal in a leading article on Increasing Muscle Strength[9] points out that the effect of training is to improve neuro-muscular adaption. This implies better co-ordination and greater economy of physical effort. Static exercises of increasing intensity produce an increase in motor unit activity, reduce synaptic resistance, improve pro-prioceptive facilitation, increase the number of pathways to the anterior horn cells and bring in the aid of neighbouring muscles. It points out the fallacy of the belief that weight lifting and heavy resistance exercises may slow down the reflexes of athletes.

Bannister[10] pointed out that the physiologist and the athlete often approach training with different aims. The physiologist, he suggests, may be able to help in the selection of athletes and in the measurement of the limiting factors, such as the control of breathing, which may affect athletic performance.

Innate properties of muscles, such as muscle viscosity, are not influenced to any great extent by training. On the other hand training can select separate muscle qualities for development. The weight lifter performs static exercises to develop force of muscle only; the discus thrower and shot-putter must produce speed as well as force of muscle and their training includes both static and dynamic exercises.

Bannister states that the greatest scope for athletic improvement lies in the distance events. Here the blood sugar concentrations must be maintained and, when fatigue has set in, neuromuscular co-ordination of a series of intricate movements may help the athlete to excel in his own particular sport; yet he might be entirely unable to master the series of movements required for another sport.

Bannister goes on to explain that the main limiting factor in athletic performance is lack of oxygen, and that in training for distance running the athlete conditions himself to oxygen lack. He also points out that all unnecessary muscular tension must be replaced as far as possible by controlled relaxation, and that the study of training methods is helpful both to the physiologist and the anatomist.

a. *Acclimatisation*

If the athlete understands beforehand what type of stresses and strains he is likely to meet in overseas meetings, such as the Olympic Games, or during a strenuous cricket or football tour, he is more likely to stand up to them. Long journeys by car, motor coach or air, with alterations in the times of meals, type of meals and sleeping routine, can be most upsetting. It is therefore of great benefit for an athlete or a team to arrive some days in advance.

b. *Training for international competition*

Reaching a peak of training in a foreign country may be difficult for an athlete or a team. The facilities he needs may not always be readily available. The wise captain or experienced team manager should make the necessary preparations in advance.

c. *Psychological factors*

There is no doubt that emotional factors are of the greatest importance. Concentration at the right time combined with relaxation afterwards is only accomplished by continuous training in the months before the event. Some athletes are able to sleep between each effort, such as their turn in putting the shot, pole vaulting or high jumping. The great batsman at cricket or the champion tennis player must develop powers of concentration of the highest order. Franz Stampfl considers these psychological factors to be vital to the first class athlete.

Bannister[11] concludes a discussion on muscular effort with the following words: 'Yet the difference between athletes lies not entirely in difference of cardiac output or diffusion capacity; it lies rather, I suspect, in their capacity for mental excitement, which brings with it an ability to overcome or ignore the discomfort—even pain—in muscles and the brain which is probably cased by ischaemia and the consequent changes of blood lactate concentration and pH. Though physiology may indicate respiratory and circulatory limits to muscular effort, psychological and other factors beyond the ken of physiology set the razor's edge of defeat or victory and determine how closely an athlete approaches the absolute limits of performance.'

d. *The avoidance of simple complaints*[12]

Muscle cramp: This is a very unpredictable condition which may be due to a neuro-muscular upset. Muscle cramps in the legs may be due to loss of salt and fluid, and to alteration in the ionic concentration of the blood. In hot weather particularly, the athlete must pay careful attention to his fluid and salt intake. Some muscle cramps are alleviated by the administration of quinine hydrochloride or sulphate tablets gr. 5, one tablet morning and night for a short period; or sodium lactate tablets 10 grains t.d.s. It would be interesting to know whether the ordinary stitch can be relieved by spinal manipulation or whether its cause can be attributed to some reflex neuro-muscular upset in the dorsal spine.

Stitch: This common complaint is thought to be due to tension on the peritoneal suspensory ligaments of the stomach. It is therefore often caused by eating too large a meal immediately before taking part in an athletic event.

In some cases the structures constituting the anterior abdominal wall over the epigastric region become tender to touch. Often the condition can be cured simply by avoiding a heavy meal within two hours of taking part in strenuous exercise.

The occurrence of foot blisters, sunburn, primary dehydration with electrolytic loss of sodium, can usually be avoided by simple methods.

Suitable clothing is of vital importance in cold weather; almost all athletes now wear track suits to keep themselves warm between events. Such attention to detail helps to produce that extra 1 per cent of efficiency which makes for champion performances and reduces the incidence of injury.

A team going on an extensive tour should have medical care arranged beforehand; a physiotherapist with an intimate knowledge of athletic injuries should travel with the team, and the players must be made to realise that immediate treatment reduces the effects of injury fourfold.

4. TRAINING AFTER INJURY

After an accident, exercises must be carefully graduated, starting at the end of the second or third week. The late Rowley Bristow used to say that if an athlete tore a muscle such as a hamstring, in the middle of the season, it was unlikely that the injury would have recovered sufficiently to allow him to continue to play for the remainder of the season; but today modern methods of treating moderately severe muscle injuries and moderately severe joint injuries make it possible for the athlete to return to vigorous sport in two to six weeks.

A tear of a muscle, such as a hamstring, produces a tender thickening which, if left to develop, organises into a tender scar adhering to surrounding structures. The power of the muscle may be greatly reduced. The products of bruising may spread down the leg and form further tender thickenings with adhesions in relation to the calf muscles, and consequent shortening of these muscles. Today, with appropriate treatment the patient may be free of symptoms by the end of the second or third week. If he is wise, however, he will not rush back to full activity without conditioning himself again. After his restoration to health, as it were, he needs to get fit. This process may take a further two to three weeks, so that in a muscle tear return to full activity may be possible between four to six weeks. Many athletes who tear a muscle do not avail themselves of these modern methods and often do nothing but rest. After a fortnight, thinking that they are fit enough, they return to the game with the injured part strapped; only to find after a few minutes or towards the end of the game when they are tired, that the muscle tears again.

If properly treated, a muscle injury should be well enough for ordinary use, that is to say about 80 per cent recovered, by the end of the second week. There should be no limitation of movement or pain in carrying out the ordinary actions of every day life. Full restoration of the muscle should take place within a further two to three weeks, if the patient undertakes gradual training as though he were preparing for the beginning of the season and also performs special exercises for the injured limb. When the torn muscle is one of the hamstrings, short, sharp movements should be avoided for the first week; but special stretching exercises, such as touching the toes and exercises against resistance give good results. Swimming and bicycling are particularly good for a hamstring tear because they develop power without full weight-bearing.

Moderate joint sprains similarly need energetic treatment. So many athletes simply rest: they often do nothing much about home treatment: they may apply a bandage of sorts and rest the joint from strain; but when they try out the joint again in two or three weeks they are unaware that it has not recovered full range of movement, and consequently, as soon as they start to play they find that it is painful, or that it is weak and tends to give way. A moderate sprain of a joint, even a second degree sprain in which there has been a synovial effusion should with proper treatment have recovered enough by the end of the second week for the patient to start graduated training.

At the Southampton Football Club[13] the policy has been 'Treat and Train'. The injured part is treated as many as three or four times a day by a physiotherapist specially trained in athletic injuries. At the same time the training programme goes on so far as it can. If, for example, the injury is to the leg, the rest of the body is kept in training. The arms, neck, shoulder girdle and back are exercised so that at no time do the muscles lose their efficiency. As the injury recovers so the right amount of specific exercise is performed.

An athlete, while recovering from an injury to one part of his anatomy, may be in a position to perfect a manoeuvre in his particular game which could have an important bearing on his team's efficiency. For example, one of the British Lions Rugby Football Teams touring South Africa in 1955 sustained a fracture of the carpal scaphoid (hand) and while it was healing in a plaster of Paris cast he made himself into a first class place kicker by constant practice for many hours each day.

Conditioning an injured limb: Sometimes it is possible, while part of a limb is in a plaster of Paris cast for a fracture, to carry out isometric exercises against the resistance of the plaster. The ends of the fractured bone can be subjected to increasing degrees of jarring and impaction forces. For example,

a noted cricketer fractured one of the metacarpal bones of his left hand. When in a plaster of Paris cast he subjected the fractured hand to increasing impactilio by holding a bat in the fractured hand and tapping the bat against a fixed object, such as a table or door. As soon as the cast was removed he could subject the hand to jarring strain to which it would otherwise have taken weeks to become accustomed. Often too when a steeplechase jockey fractures a clavicle he is taught how to break the strain on the shoulder girdle by fixing the elbow and shoulder joints so firmly that little strain is thrown on the fractured clavicle. This is the reason why many steeplechase jockeys can return to race riding in a fortnight.

With the use of hydrocortisone, hyaluronidase, other forms of injections, aspiration and expression of excessive exudations, recovery from athletic injuries, especially those which are accompanied by excessive contusion, can be considerably accelerated. But the need for both home treatment and organised physiotherapy is not lessened by these special methods. The athlete must carry out his part, remembering always that attention to detail is one of the most important factors in regaining maximum efficiency.

REFERENCES

1. Hill, A. V. 1951. The Mechanics of Voluntary Muscle. *Lancet* 1951, Vol. 2, pp. 974-951.
2. Coombes, M. E. and Pugh, L. G. C. *Pre-Ski Exercises*. Ski Club of Great Britain, London.
3. Anthony, Don, in Collaboration with the Fitness Advisory Bureau. 1961. *Keeping fit for all ages*. Published by Educational Productions Ltd.
4. Alford, J. W. L. Personal communication.
5. State, Oscar, 1955. *Weight training for Athletics*. The Amateur Athletic Association, London.
6. MacQueen, I. J. 1956. Progressive resistant exercises for athletics. Lecture to British Association of Sport and Medicine, London.
7. Bleasdale, H. N. C., 1960. The controlled interval method of training as applied to swimming. Lecture to British Association of Sport and Medicine, London.
8. Stampfl, F. 1957. Slavery of Athletics, Mind and Body, How Far can a Man Go. Three articles appearing in the *Observer*, July/August, 1957.
9. *British Medical Journal*. 1957. Increasing muscle strength. Leading article 1957. Vol. 2, p. 150.
10. Bannister, R. G. 1955. Athletics and Physiology. Lecture to British Association of Sport and Medicine.

11. Bannister, R. G. 1956. Muscular effort. *British Medical Bulletin.* Vol. 12, No. 3. Physiology of Voluntary Muscle, pp. 222-225.
12. Abrahams, Sir A. 1954. The heart in athletes. Lecture to British Association of Sport and Medicine.
13. Featherstone, Donald F. 1954. A symposium on athletic injuries and their treatment. Lecture to British Association of Sport and Medicine.

5

THE SIGNIFICANCE OF SOMATOTYPE IN SPORT AND ATHLETICS

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WHEN considering the significance of somatotype in relation to any group of people, we ought to bear in mind that there are as many different kinds of human being as there are human beings. This is a generalisation which is confirmed rather than shaken by a closer study of methods for classifying human physique and behaviour. It should be remembered by those who are seeking new and improved methods of selection and training for sport and athletics. The report on the 1952 Olympiad by Professor Jokl and his co-authors entitled 'Sports in the Cultural Pattern of the World',[1] has shown convincingly how frequently an athlete has succeeded in overcoming all environmental and constitutional adversities to beat more favourably endowed athletes. Reaching the acme of physical endeavour is a very personal achievement, and the possibility of an occasional athlete excelling in an event for which scientific somatotyping may deem him, or her, constitutionally unsuitable, must not be disallowed.

In 1921 Kretschmer[2] described three types of physique: asthenic, sthenic or athletic, and pyknic. Obviously all varieties of human physique do not conform to these three limited types. Sheldon[3] has replaced this narrow classification by one that is wider and based on body shape rather than body size. In Sheldon's classification each individual may be placed and described quantitatively in terms of three components of body structure. Parnell[4] has given a concise account of this method. The physical properties described by each of these three components are as follows:—

1. *Endomorphy:* describes roundness of physique and capacity to store fat.
2. *Mesomorphy:* indicates the degree of bone and muscle development.
3. *Ectomorphy:* portrays the linearity or slimness of build. It is closely related to the Ponderal Index—height divided by the cube root of bodyweight.

Each of these components is rated on a seven point scale and the combination of the three numbers so obtained for each component labels the individual somatotype. Different types can be schematically related to one another

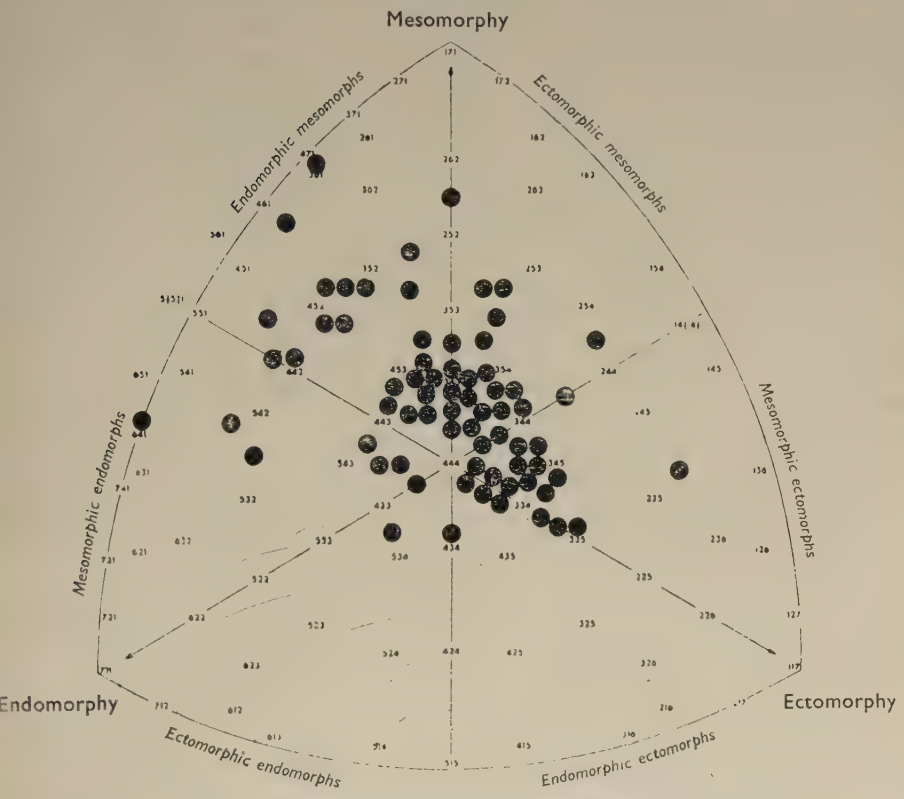

FIG. 1. Diagram showing a schematic two dimensional projection of the theoretical spatial relationships among known somatotypes.

by plotting them on the Somatochart (Fig. 1). At the centre of this chart is somatotype 444 (i.e. a rating of four points each for endomorphy, mesomorphy and ectomorphy—4 being the mean figure between 1 and 7 on the seven point scale) and represents the balanced body proportions of the theoretical average man. The fat endomorphs lie southwest of this mean point, muscular mesomorphs lie to the north, and the thin Ectomorphs can be plotted to the south-east. These three groups can be subdivided according to dominance of ratings in the remaining two components. Thus endomorphs may be more muscular than average for endomorphs, mesomorphs may be more fat or thin than average for mesomorphs.

There are several methods by which these components, fatness, muscularity and linearity can be assessed for any individual:—

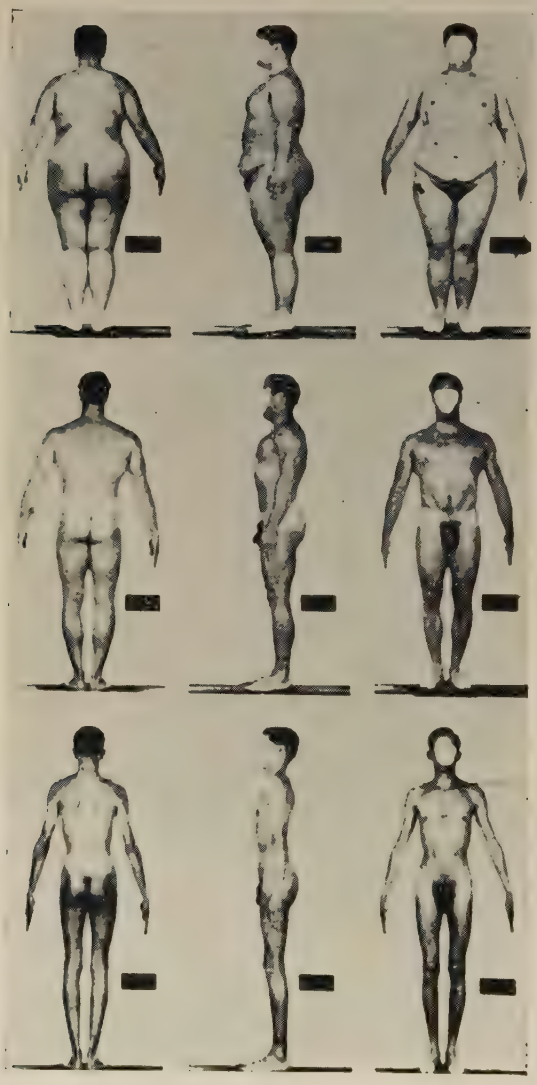

FIG. 2. Standard somatotype photographs showing examples of endomorphy, mesomorphy and ectomorphy.

1. *Scopically*, by studying standard photographs.

2. *Metrically*, by taking photographic measurements and referring to Sheldon's tables.

3. By a combination of 1 and 2.

4. *Parnell's simplified method* by physical measurements and reference to the Standard Deviation Chart. This method is best if supplemented by Sheldonian standard somatotype photographs.[5]

Figs. 2 and 3 show Sheldonian standard somatotype photographs of extremes of endo, meso and ectomorphy and also a mesopene. (Fig. 3.) The mesopene is a particularly interesting type. He is a 'fat-thin man' with poorly developed bone and muscle. Such types rarely distinguish themselves in any sport, and usually have no inclination for exercise.

Of special interest to those concerned with the training of athletes is the relationship which appears to exist between physique and temperament. In an extensive study Sheldon has been able to link somatotype dominance with dominance of certain character traits.[6] Parnell has also made valuable con-

FIG. 3. Standard somatotype photographs showing the mesopene (the 'fat thin' man).

tributions to this aspect of somatotyping.[7] Sheldon describes three main character types, each related to a particular somatotype dominance. They are:—

A. The '*Viscerotonic*' personality of the endomorph. Genial and expansive, easy-going and a lover of comfort, he shuns exercise. Fond of social wining and dining and tends to live to eat rather than eat to live.

B. The '*Cerebrotonic*' personality of the extreme ectomorph. This is in direct contrast to that of the endomorph. Although often of voracious appetite he eats only to live and derives little pleasure from social dining. Quiet, shy and retiring he avoids crowds, brightly lit rooms, tends not to make

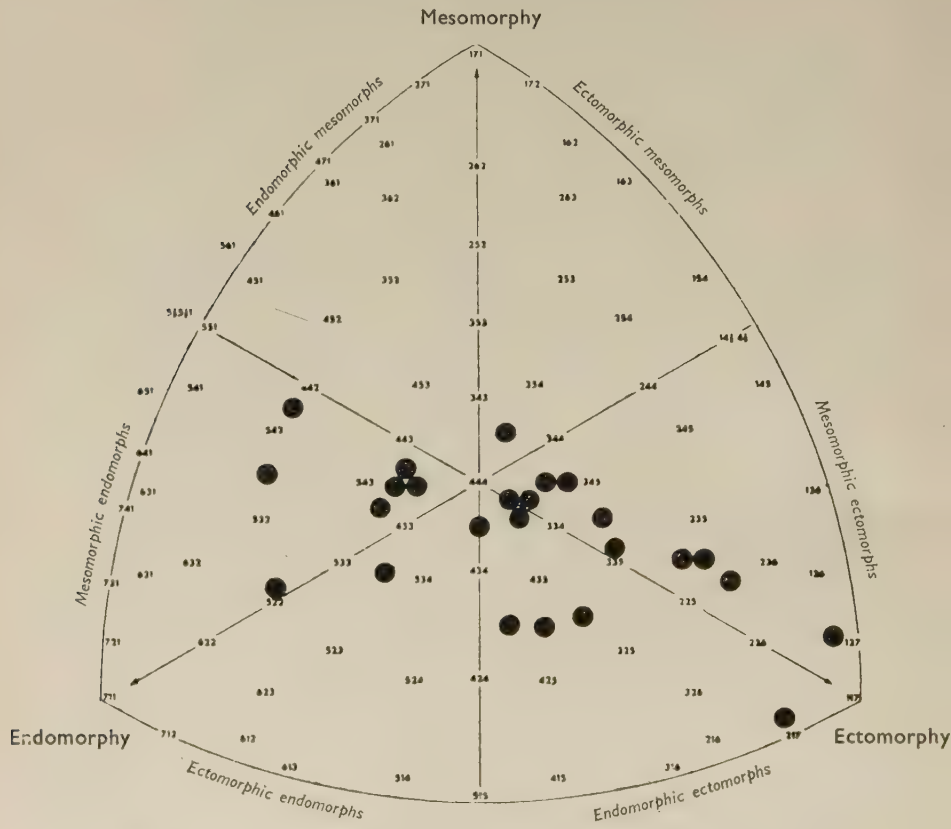

FIG. 4. A schematic two dimensional projection of the theoretical spatial relationships among the known somatotypes of those who take no exercise and who have no inclination for vigorous pursuits.

friends easily and is introspective. He shrinks from violent exercise and regards any display of physical strength as something bestial.

C. The '*Somatotonic*' personality of the mesomorph is characterised by a Spartan attitude to life, spurning physical comfort and thriving on exercise. He is often aggressive with a love of combat and adventure.

Fig. 4 shows how the somatotypes of those who take no exercise and have no inclination for it nearly all fall below the mean rating of 4 for mesomorphy. Men with a mesomorphic rating of less than 3 participate in sport less often than those having greater muscularity, and participation in the rough contact sports is rare. Conversely, those who enjoy strenuous outdoor exercise are grouped with a decided dominance of mesomorphy. (Fig. 1.)

These wide variations in human physique and temperament, present among

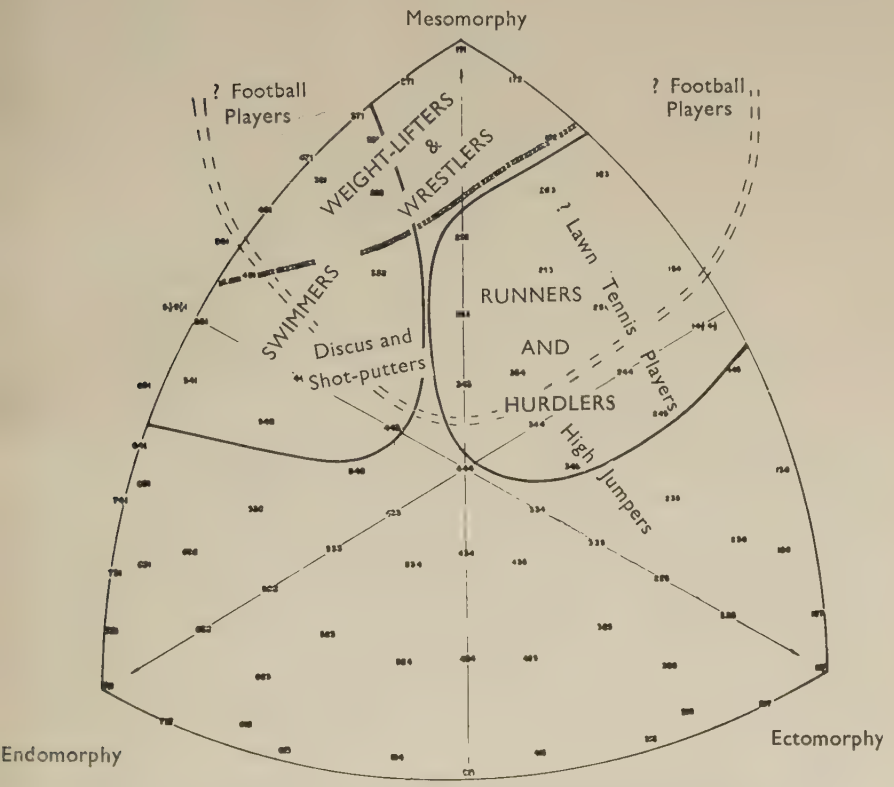

FIG. 5. A schematic two dimensional projection of the theoretical spatial relationships of various types of athletes.

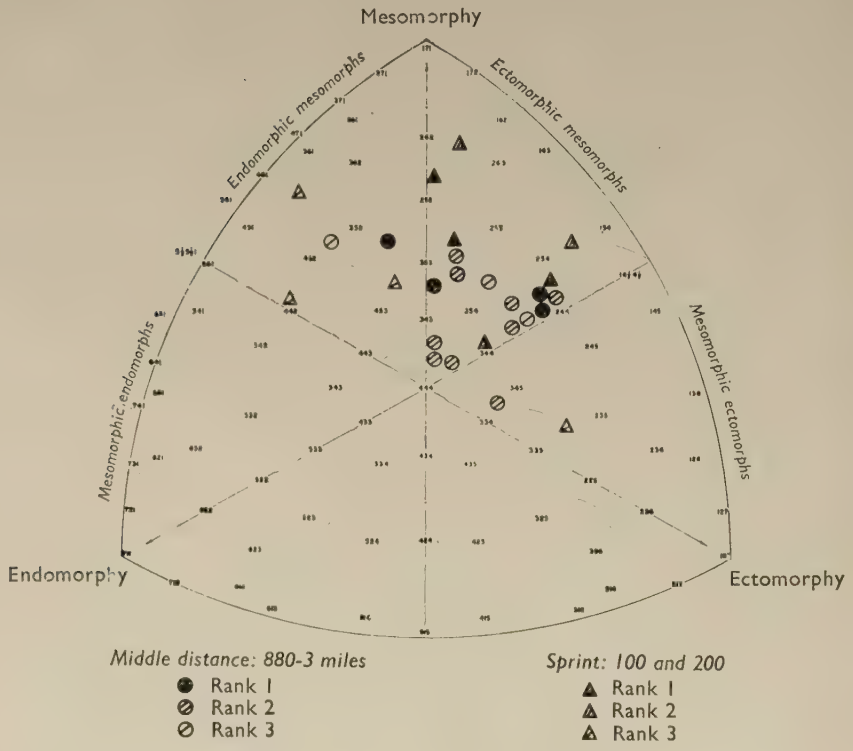

FIG. 6. A schematic two dimensional projection of the theoretical spatial relationships of runners of various types.

athletes no less than among non-athletes, suggest that an attempt should be made to recognise an individual's athletic potential and aptitude early by scientific somatotyping. Training should then be designed to suit the constitutional needs of the individual. The method could possibly save wastage in the training of men for events for which they are constitutionally unsuited, and conversely, help to ensure early recognition of a man's capacity for a certain type of physical endeavour.[8]

A study of the somatotype distribution among different classes of athlete (Figs. 5, 6 and 7) shows how most groups have a rating of 4 or more for mesomorphy. Swimmers and wrestlers have a higher fat component than runners, hurdlers and jumpers, i.e. swimmers and wrestlers tend to be endomorphic mesomorphs while runners, jumpers and mountaineers are ectomorphic mesomorphs. Throwers, of discus, hammer and shot, and pole vaulters are

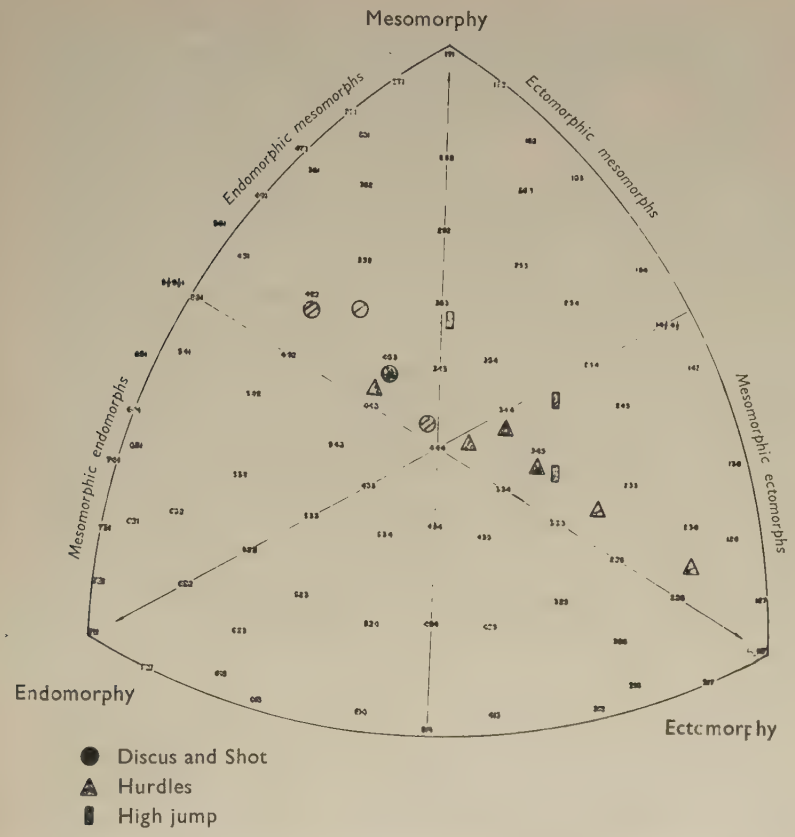

FIG. 7. A schematic two dimensional projection of the theoretical spatial relationships of other types of athletes.

also fairly strongly mesomorphic with an endomorphic rating between 1 and 3.

There is an interesting variation in the somatotype rating between runners of different distances. Thus long and middle distance runners frequently show ratings of 245 or 234, whereas sprinters tend to show higher muscular and lower linearity ratings. The fat component is seldom higher than 2 in either group. This reflects the need for longer legs and lighter bodyweight by the distance runner who has to maintain highly repetitive movements for a long time, and the need for a sturdier, more muscular physique by the sprinter who has to make an explosive effort for a short time only.

Ectomorphs with a muscle rating of less than 3 may do well at games of skill like tennis, table tennis and golf and some make good walkers. That sec-

tion of the non-athletic world which is predominantly fat with little muscle has a too inadequate power/weight ratio for effective participation in any strenuous physical activity. In this respect the Mesopenic 'fat-thin man', or endomorphic ectomorph, is at a particular disadvantage, both constitutionally and psychologically.

FIG. 8. Two international packs—there can be no doubt that individual somatotyping of these forwards would show remarkable similarity.

Reflecting on all this scientific data an athlete could well be excused if he came to the conclusion that he should adopt a fatalistic attitude, and resign himself to training and participation within the narrow field of sports dictated by his somatotype rating. But the opening paragraph in this chapter should dissuade him from this attitude. The need of specialised training techniques to suit individual variations in physical constitution has long been recognised by weightlifters. They practice a system of what they call 'type training' which involves quite complex training schedules designed to suit the particular somatotype dominance of different lifters.[9] The underlying principle of 'type training' is the employment of a 'heavy and light' system of exercises.

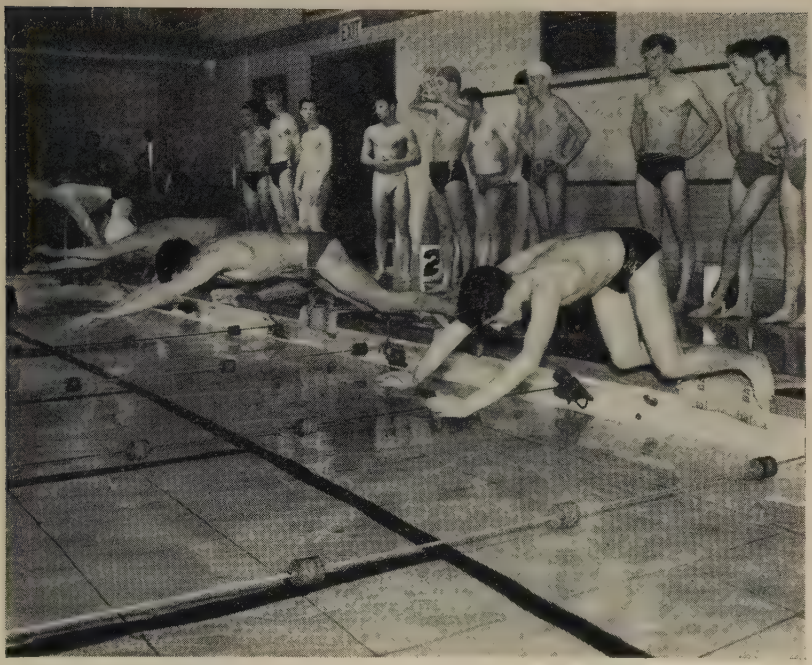

FIG. 9. The start of a relay swimming race—would somatotyping help to pick out potential champions?

In its simplest form this means that short periods of low repetition heavy resistance work are combined with highly repetitive low resistance work. The proportions of heavy and light exercise are adjusted to suit individual temperament, physical constitution and choice of athletic event. A simple application of this principle is seen in a boxer's training. Heavy bag punching and road work is combined with light skipping and footwork. In preparation for the first under-four-minute mile Dr Roger Bannister ran serial quarters of a mile at all out speed, but also ran longer distances than a mile at a more leisurely pace.[10] This is another example of the practical application of heavy and light training. An increasing number of championship athletes are incorporating some form of heavy and light training in their workouts. Weight resistance exercise offers a convenient way of applying this technique and is frequently used. Several refinements of this technique exist and perhaps one of the most useful to athletes is the 'velocity training' technique. Before trying to increase the weight used in any exercise the *speed* of exercising is increased

until the given number of repetitions is performed in minimal time. Only then is any increase made in the amount of weight resistance, and the procedure repeated.

Too little work has been done, so far, concerning injuries in sport and athletics according to somatotype to draw any definite conclusions. There is some evidence to suggest that the fatter mesomorphs sustain more fractures and sprains, although these injuries are not always sustained at sport.[11] It must be borne in mind that mesomorphs tend to take part in strenuous contact sports more often than do endomorphs and ectomorphs, and therefore, a higher incidence of injuries is to be expected.

REFERENCES

1. Jokl, E., Karvonen, M. J., Kiklberg, J., Koskela, A., Noro, L., 1956. 'Sports in the Cultural Pattern of the World'. Institute of Occupational Health, Helsinki.
2. Kretschmer, E., 1921. 'Korperbau und Charakter'. Springer, Berlin.
3. Sheldon, W. H., Stevens, S. S., Tucker, W. B., 1940. 'The Varieties of Human Physique'. Harper, New York.
4. Parnell, R. W., 1954. The Medical Press, 231. No. 6007.
5. Parnell, R. W., 1954. Am. J. Phys. Anthrop. n.s. 12, No. 2, 209.
6. Sheldon, W. H., Stevens, S. S., 1942. 'The Varieties of Temperament'. Harper, New York and London.
7. Parnell, R. W., 1958. 'Behaviour and Physique'. Edward Arnold Ltd., London.
8. MacQueen, I. J., 1957. *Journal of Physical Education*, 49, No. 148, 79.
9. MacQueen, I. J., 1957. 'Type Training'. *Health and Strength Annual*, Health and Strength Publishing Ltd., London.
10. Bannister, R., 1955. 'The First Four Minutes', Putnam, London.
11. Parnell, R. W., Personal Communication.

6

PHYSIOLOGICAL AND PSYCHOLOGICAL APPROACH TO ATHLETICS

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THE IMPORTANCE and value of competitive sport in a comprehensive system of education has long been recognised both for the obvious reason of the promotion of physical fitness and for its part in the development and building of character. Competitive sport teaches and accustoms the young man or woman to physical discipline, to self control, which is in reality only self-discipline, and the vitally important art of getting on with other people.

Physical and intellectual education are at once competing and complementary. A state of underdevelopment results if there is not a proper balance between the two, a fact that tends to be missed as the pace of life increases. Many people in responsible positions overlook the very practical fact that the social side of sport provides an obvious opportunity of mixing socially with people of vastly different backgrounds, to the advantage of their work and the broadening of their personal outlook.

One may consider that people fall into three classes. At one end of the scale is the student who devotes the whole of his time and effort to learning his trade or profession and who is so often encouraged to do so by academic men who have themselves almost lost contact with the practical living world. At the other extreme are those who give all their time to sport and look on study as something that interferes with the more important things of life. They frequently have more than their share of the social graces and are even popular with their harder working contemporaries, but for some reason just cannot get down to learning a trade. In between these two groups are the large though rapidly shrinking number who have learned to work hard and play hard, giving roughly equal attention to each. Of course there are no sharp boundaries between these groups and it is really better to think of them as a continuous spectrum extending from 'All work and no play', through the properly balanced, to all play and no work, either of which makes Jack a dull and imperfectly educated boy.

Many are able to satisfy their need for sport with field sports and other only very indirectly competitive pastimes. There are, for example, rather surprisingly more subscribing members of angling societies in this country than of football clubs. It is with athletics, however, that we are mainly concerned in this paper and it is to the factors which contribute to the enjoyment and success of the competitor in these events that we shall now turn.

Thousands of young men actively take part in sport every week and of these some will succeed but only a few will excel. This small minority are the elite, the star athletes who by their prowess will bring honour to themselves and prestige to their country. Analysis of the reasons for this super quality is difficult and complex, for physically they represent all sizes and shapes. Some will immediately impress by their athletic bearing but others remain a constant source of surprise, since they are small, ill-formed and of poor physique. A few will have physical defects which should reasonably prevent them from active sport. All, however, have a natural talent for their sport, physical strength and toughness and dedication; without this basis it is difficult to see how the acquired skills can be fashioned to give these star qualities, nor can it be accepted how any human can withstand the programme of training which they have built up without dedication and devotion. However, they have one asset which is common to all and that is sensitivity above the average. This gives them psychological and physiological differences and is characterised by an increased sensitivity of mind and body. They bring to sport both the high qualities and defects which characterise them. These men are highly conscientious and make every possible effort to master techniques and minutae of their chosen event. No training period is ever missed; and provided they have confidence in their coach they will follow religiously any programme that is ordered. Careful assessment of their physical capabilities is essential lest they drive themselves too far and too hard. They set a high standard for themselves and expect and require other people to do the same. They do not suffer fools gladly either in work or in play. An important attribute is that they are conscious of the people around them and so can anticipate the moves of their opponent. From this group come the leaders—those capable of the quick explosive start and the electrifying finish to a race. However, they are easily disturbed by environmental factors and petty irritation, travel badly and tend to arrive tired. As the time of an important event approaches psychosomatic symptoms tend to appear and the masseur is visited for muscular pains. With their labile emotional make-up, relaxation before an event is essential to them: and should anything go wrong with the timing and the event be delayed, restlessness and irritation lead rapidly to loss of physical and mental energy and performance suffers disastrously.

A young man likely to aspire to international standard has about eight years to prove his worth. Besides enthusiasm for and dedication to his chosen event, he requires the will to win (which is not the same thing), and the right degree of emotional stability; and if not actual economic independence, at least a sufficient sense of security to avoid having his training interfered with by financial and personal worries. The basic training for all athletes is the same and aims as much to attain the highest possible standard of physical fitness as to perfect the specialised skills of the individual event. One of the few exceptions is the sprinter. It is axiomatic that great sprinters are born and not made and it really appears that they require less training than other athletes. The middle and long-distance runners, however, have to follow a course of training and achieve a physical standard which continues to astound the physiologist. Zatopec first devised the intensive training schedule for these events which all endurance athletes now try to emulate. Every day he would cover the 400 metres track thirty to fifty times alternatively walking and running, practising intermittent breath-holding during the walking phase to accustom his autonomic nervous system and respiratory centre to high carbon dioxide tension, while gradually building up his oxygen intake to an enormously high level. It was he too who developed the practice of 'running through' fatigue. Fatigue normally results in incoordination and hence loss of performance: but by training he found it possible to master this and while running to recover to further effort.

A new conception of training is thus emerging. Prior to the second world war the recognised pattern of training was based on the principle of slow progressive advance aimed to avoid at all cost the danger of overstrain and breakdown. During the last decade, however, following the example of Zatopec, training schedules have become much more intensive: and indeed although many have closely mimicked his technique, others have added even more strenuous routines, with no apparent injury to health. All this has excited the curiosity of physiologists and is causing them to revise their views on the basic principles governing physical performance; but many problems remain to be solved before we know how to train an athlete to produce his maximum performance.

It is impossible to forecast how far the present remarkable rate of advance in athletic performance can go. Not so long ago the four minute mile, now almost an everyday occurrence, was regarded either as an impossibility or at least as the ultimate goal of athletic achievement. It is recognised that the body keeps a large reserve of strength as a protective mechanism against the risk of strain or injury from excessive demands in an emergency, but no one knows to what extent this reserve can be mobilised by modern training

methods, or what will be the result if, by more strenuous methods, this hidden reserve is drained off. Suffice it to say that it is only an exceptionally healthy individual who can undergo the stress necessary to tap this reserve without the risk of injury. Now and again an athlete appears who is prepared to push his potential resources to the uttermost and who is able to keep up the pace day after day without deterioration in health or performance. Such an athlete is a great rarity and appears but once or twice in a generation. He is at once the coach's dream and his nightmare. He can only be identified by his ability to go on where the ordinary athlete would break down, but once found and provided he and the coach get on together, they can tap his hidden reserves and many records will be broken.

Repetitive practice is an aspect of training most obvious to the outsider but which plays a comparatively minor part in modern training methods. It consists essentially of establishing a series of conditioned reflexes. At the beginning of his career a person who takes up any skilled activity from running to fly-fishing performs awkwardly and has to think each move and the position of various parts of his body. By repetition he gradually masters the complicated series of movements needed and stores them in his subconscious mind and as conditioned reflexes in his pyramidal and extrapyramidal motor system—a chain of pathways in the brain which can initiate the movements effortlessly and co-ordinate action when required. In driving at tennis for example, only the decision as to what part of the opponent's court is most vulnerable, is likely to be taken at conscious level; the technique of driving is looked after by a series of conditioned reflexes involving the pyramidal motor cortex, and the accurately co-ordinated pattern of muscular contractions required to hold the racket in the correct driving position is initiated and maintained by the rubro-spinal system. Similar factors are at work in such athletic events as throwing the hammer and putting the weight. However, there is a limit to the purely technical benefit of repetitive practice, and over-familiarity may result in loss of performance from failure of concentration, the whole procedure having in effect sunk to the reflex level. Training in concentration is just as important as mastery of the technical skills. Agility, the ability to react quickly and precisely to a sudden stimulus, as in a catch at slips, is one of the most impressive features of the trained athlete and especially in sprinting and field events, a major requirement and one largely acquired through repetitive practice. The shot putter and hammer thrower, who have to work up speed in a small circle, often seek to improve their agility by running 25-yard sprints against the stopwatch.

The part played by the coach in the selection, training, and management of the modern athlete is now beginning to emerge. 'A promising young 'un

does not often make a champion old 'un,' and the ability to recognise potential Olympic material at an early stage is one of the most hardly acquired abilities of a good coach. The age of physical maturity varies widely, and of mental and emotional maturity even more so; indeed many young men in our artificial society never achieve it and pass from adolescence to middle-age and senility without ever having passed through a phase of life that could be described as their prime. The potential 'great athlete' can be 'burnt out' and ruined by ill-advised overtraining just before the correct time, and only great experience and the expert study of the individual can enable the coach to recognise the point at which intensive training can safely be undertaken. The wise coach will make no firm promises for the future and will watch constantly for the first signs of staleness in a young athlete, as evidenced by early fatigue, insomnia and weight loss.

The function of a coach is not only to select and train but to inspire. A great coach can inspire an athlete, when appropriate, to train to exhaustion, to run through fatigue, and to achieve the well-nigh impossible. He can often make a merely average athlete great for a day. In the course of training, a very close mutual understanding develops between athlete and coach, and the athlete develops a degree of dependency which renders him suggestible to encouragement and reassurance, a relationship which allays fears of defeat and anxiety about strong rivals.

We have said nothing so far of the mental approach of the athlete himself; and indeed very little planned research into the psychological aspects of sport appears to have been done. It has been suggested that there may be a psychological barrier in athletes, the protective mechanism which conserves the hidden reserve of physical strength. Already it is felt that very little further can be achieved in the study and application of technical skills, and most coaches consider that they are very near the limit of physical toughness and strength. They now feel that the third approach is necessary, and that the emotional problems and psychological aspect of the athlete must be studied and corrected so that the mental toughness so necessary in international sport can reach the standard already achieved in physique and technical skill.

The need for a psychiatrist as a consultant in sport is becoming more apparent. The introduction of such a service requires careful preparations and planning, for there is a prejudice against psychiatry in the public mind since it is identified with insanity, mental defectives and ill health and may not be readily acceptable. These prejudices can be overcome by teaching what his speciality can accomplish and by educating the officials and athletes in formal and informal talks on the aims of a constructive mental health programme and, by stimulating an interest in the aims of good mental health. His role there-

fore in these early stages must be by instruction on simple physchological principles; but as he becomes more acceptable he can then provide all the skills which his speciality can offer. It would be important too to have other assets, a worldliness and a wide knowledge of affairs so that he can make best use of the social functions. It is obvious that he must have a knowledge of the game or sport, its technicalities, its subtleties, tactics and strategy so that he can talk with knowledge to the players and, more important, understand the difficulties of the various positions in the game. It would be wise also to learn the team's policies and practices, the rules that govern the group, and the attitude of the officials, so that any suggestion made will be consistent with existing custom. The coach is the lynch-pin of the team and the officials, and the relationship between him and the athlete is very close. From continual observation he knows the athlete well, his strength and weaknesses, his capacity and potential. The athlete follows closely the guidance and teaching of the coach and is dependent on him for his programme of training. Since discipline is essential in such a strenuous schedule it is necessary that psychiatrist and coach should work together. However, it must be firmly established that much of the information gained by the psychiatrist must remain secret since, while he may be able to cure some defect by simple means, others may require analysis into intimate matters and reports on these must be in general terms only.

This new approach to sports opens up a fascinating field of research and the results and conclusions can be of enormous help in assessing and understanding the emotional needs of the athelete. (It must be granted that the intensive training and competition needed to succeed in modern sport is an excellent screening and the final selection by this method ensures sound, well-balanced personalities.) With the increased tempo of life, the competitive world, noise and speed of modern life, many sportsmen are near the limit of endurance. Many doctors whilst astonished at the capacity of men to withstand such training, are rather apprehensive about the eventual result. All help therefore should be given them so that they may understand the mental and physical strain involved and how to combat them.

But this is part of the athlete's life which is known. Private life, which the public do not consider and which is often forgotten by knowledgeable people, can hold stresses which will cause poor performance and indifferent form. Hans Selye has shown in animals, that persistent exposure to a particular stress increases resistance to that stress but at the same time it reduces resistance to other kinds of stresses. This applies particularly to sportsmen, since an athlete who has trained his body to muscular exercise will be able to withstand the stresses arising from severe exercise but will have decreased resist-

ance to other stresses encountered in the usual pattern of life and these can hasten a breakdown. This can happen to a few after a minimum of stress but in others only after a severe and prolonged test. All living organisms have a limit to the amount of stress that can be tolerated since adaptive energy in everyone is limited. In the trained sportsman, breakdown is usually mild and could be called staleness, and is usually followed by quick recovery. The type of sport has some effect on the problem. Team games with sharing of effort and of disappointment, the interdependence of members on each other, can delay the effect and keep morale going whereas sports requiring individual effort and responsibility are a much greater strain.

Staleness can be due to some inherent fault, but other factors contribute. Training occupies much of the day, so that economic and social security is essential; and unless the athlete can attain it, it would be better if he gave up sport. It is readily understandable that bereavement in the family, loss of a friend, family conflicts, girl friend trouble, studying for examinations, failure to succeed, are stresses; and if the test is too severe, they can cause breakdown and the finish of a man's sporting career. It must be emphasised that the athlete has no insight into his state of health in the early stages and blames everyone and everything for his loss of form but never himself. Early diagnosis is most important. The early signs and symptoms are loss of appetite, loss of weight, tiredness, heaviness of the legs, pains in legs and joints, irritability and sensitivity, feelings of resentment, lack of interest in training, the desire for peace and quiet rather than competition, withdrawal from and avoidance of people and resentment if he is questioned on his own or his team's performance. He may consider giving up sport altogether. Rhinitis; skin rashes, boils and pimples are common. The coach and officials will be well aware of this change in temperament and must take firm action by stopping all training and competition for some time and seeing that rest and sleep—induced sleep if necessary—are assured. Later other treatment may be necessary, and it is at this stage that the psychiatrist can help by evaluating the cause of the trouble and restoring his emotional state to normal.

Mental health consists of inherited and acquired potentialities all integrated and working to a common end for the good of the individual and the proper function of his whole personality. Whilst there is little control over the inherited factor, the acquired potentialities depend on upbringing, the background of the home and the teaching and training that moulds the young life. In this way ideals are formed and life becomes purposeful and directed to a common end. Since we all have different capabilities, this permits a wide range and variety of personalities. The principal characteristics necessary to mental health are discipline, self reliance and independence. Under present

day circumstances these are the most difficult to develop. The Welfare State with its all-embracing protectiveness leaves little room for many individuals to acquire independence and reliability and makes life so secure that these important characteristics are not necessary for living. The first essential for mental toughness is mental health. Here the psychiatrist will have great scope for his training and skills and will have made a major contribution to the athlete and sport if he can solve it. That this important state can be developed is shown by the experience of World War II. The Services, to harden the soldier to war, organised a series of repetitive exercises which closely resembled the conditions that might prevail in combat. Preliminary training was to accustom men to noise of battle, that most disturbing and frightening aspect of any war. This was followed by initiative tests, hardship tests and night attacks, all carefully planned to harden and toughen the soldier, mentally and physically. It is unlikely that in sport such tests would be practicable, but there is a wide range for some ingenious and inventive person to organise and implement a scheme which would develop this essential quality in training so that mistakes and failures would not be made in actual competition. Mental toughness means greater efficiency in a crisis and when the game is tense and critical—sticking to an opponent so that he has to fight to win or crack and lose, keeping one's head and holding oneself or one's team until the right moment to strike. These qualities are seen in the experienced sportsman when he is athletically old. How much better if they could have been developed when he was young and then we could have the 'old head on young shoulders'.

Mental training must go hand in hand with physical training, for without it all the painful preparation for a big event can be lost by a false start—a dropped baton, a scramble in front of goal, etc. It must be accepted that the psychiatrist has a limited use and many of the problems submitted to him will not be resolved and are not capable of solution. However, in this neglected branch of sport, he is an essential person; and it is hoped that with his help and advice further advances will be made on research into human endeavour and the fascinating problem of the limits to human endurance.

7

WOMEN IN SPORT

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HISTORIANS have recorded the participation of women in games and sports for many centuries, but during the nineteenth century physical exercise was widely thought undesirable for women. This was in accordance with the predominance of the male in affairs of the world, in business and in home life. Muscular development was thought inappropriate for the female and athletic sports considered to lead to lack of femininity, physical strain and interference with the reproductive function. Early marriage and multiple pregnancies made girls disinclined to play games.

As women have emerged to equality with men in most pursuits of the mind, so they have shaken off many of the physical restrictions of Victorian times. The fears of ill effects from sport so freely entertained in the last century have proved groundless. Apart from menstruation, parturition and lactation, there are no essential differences in the physiology of male and female; and the special anatomical characteristics of the female offer no bar to athletic sport.

PHYSICAL BENEFITS ON INDIVIDUAL AND RACE

There has been a marked improvement in the health and physique of Western women since the first world war and this has been noticeably maintained in the last fifteen years. There is a striking advance in the physique of the girls attending ante-natal departments today compared with thirty years ago. Advance is also apparent in the girls of higher social class coming from schools at which organised games have long been established.

There is still a marked contrast between the physique of Western women of the educated classes and those from Eastern countries where women have only recently emerged from the harem. Jokl[1] lays stress on this when he states that, of sixty-nine countries sending representatives to the 1952 Olympic Games, twenty-eight sent no women. At Melbourne in 1956 India entered only seven women, Israel four, and the middle east countries none.

FIG. 1. There is nothing half-hearted about these girls' approach to athletics. It would be hard to imagine a closer finish.

While performance in world athletics affords a yard stick of progress, it is only of limited importance. It is the widespread indulgence in sport by growing girls, and women that will bring lasting benefit to the nations. Record breaking makes headlines, but it is the weekly participation in games by millions of women in the Western world that matters. Women of good physique are better able to carry out the daily tasks of life.

Enquiry into girlhood habits in regard to games and physical exercise while taking the medical history, together with a general examination of a very large number of young women during early pregnancy, has been instructive. A few were well-known athletes, many indulged in sport at school and after, but many had stopped when they left school. Strangely it is not infrequent for a girl of fine physique to say she has no interest in sport but further enquiry often reveals that she goes hiking or swimming or cycling.

The posture of the game-playing girl is noticeably better. Since man adopted the erect posture, maintained by the spinal curves, there have developed a collection of ills, probably unknown to our arboreal ancestors.

Low backache is so frequent among women that it is almost an expected complaint of those who have borne children. The correct posture taught to athletes and the muscular power developed to maintain it will help to eliminate excessive lordosis and sacro-iliac strain.

Competitive sport for both sexes favours the growth of an equable temperament and self-confidence and dispels to some extent fear of unknown experiences. The intermingling of men and women in sports helps to eliminate the 'mysteries of women'.

Women now engage in most of the games and sports open to men. Games in which bodily contact and hard knocks are an essential part are best avoided, as they may bring damage to face and breasts. There is no need for women to play football or box or wrestle. Black eyes, swollen noses and cauliflower ears don't do much harm but are certainly unfeminine. The female frame,

FIG. 2. It is doubtful if this girl's grandmother would have believed it possible for a woman to jump as far as this, but this competitor certainly seems to be none the worse for her activity.

being lighter, less muscular and often less well-controlled, is more vulnerable to minor strains of tendons and ligaments. This is confirmed in the report from the American Amateur Athletic Union.[2] Graduated training is as essential for women as for men.

GENERAL MEDICAL CARE OF THE WOMAN ATHLETE

Medical examination before embarking on an athletic career should be routine and all the usual tests of body efficiency should be carried out. Athletes should be informed of the necessity of a regular check-up, particularly after any infection. Doctors well-versed in the problems of athletes should be available and have access to clinics where the required investigations can be carried out. These should include pulse rate, blood pressure estimation and determination of the recovery of blood pressure and pulse rate after exercise. A blood count, urinalysis, radiography of heart and lungs and electro-cardiography are advisable. Gunnar Malmstrom[3] states that many athletes think they have heart trouble but few show evidence of organic heart disease. He found that extrasystoles and precordial pain were the commonest symptoms. Mental and physical stress are responsible, but it is particularly important in women that real heart disease should not be missed. Women showing a tendency to hypertension had best avoid strenuous sport.

A pelvic examination is advisable for those reporting any gynaecological symptoms. Parous women in particular should have a pre-season examination, so that if there is any evidence of genital prolapse they can exclude such activities as putting the weight, discus throwing and jumping. Ovarian cysts are not uncommon in young women. Frequently they are symptomless and are diagnosed only at the first examination after a girl has become pregnant. Obviously a girl with a dermoid cyst in her pelvis should have it removed before a hard season's athletics.

Another trouble common among young women is vaginal discharge, particularly discharge caused by the trichomonas vaginalis so frequently picked up during communal existence. Trichomonas vaginitis is accentuated by hard exercise and the irritating discharge may cause vulvitis and crural soreness. It is easy to relieve if it is recognised early, but it is liable to recur and when it is of a severity sufficient to cause secondary vulvitis the patient may require treatment in hospital.

Pruritus is a most distressing sympton, particularly that caused by monilial infection. Fortunately monilial vulvo-vaginitis clears up rapidly under appropriate treatment.

The joint committee on 'The Physical Education of Girls'[4] a committee of

medical and educational representatives, in its report in the *British Medical Journal* in 1922 was in favour of games for girls but gave warning of the danger of 'heart strain' from swimming and rowing; and certain members thought physical exertion would interfere with school work. Heart strain is a vague term and experience has revealed no evidence of any untoward effects in women from sport when the heart was not diseased. Men vary in their

FIG. 3. Women are particularly successful in swimming and diving. This girl's position shows beautiful grace and poise.

ability to continue their studies while in hard training, and the same is probably true for girls. Some work well in the evening after a hard game in the afternoon and others cannot concentrate. It is the individual's psychological approach which counts.

Year after year in athletic contests the performance of women approaches more closely to that of men. Their improvement is more startling perhaps because they have had further to go. Comparison of past results with those of today is not of great value, owing to improvements in the equipment, technique and the tracks. It is unlikely that the best women will ever beat the best men in athletics under the same conditions, or at golf off the same tees, but it is certainly true, as Jokl[1] says, that 'a well-trained woman athlete today is superior to the average untrained young man in most branches of sports'.

Since it seems that women, within the limitation of their physical development, are just as able to indulge in sport as men, it only remains for us to consider the influence, if any, of strenuous exercise upon the reproductive organs and their functions.

MENSTRUATION

Years ago almost any sport was considered unwise during the menstrual period. In normal menstruation a woman loses about 4 ozs. of blood and endometrial fragments from the uterus during the space of four or five days every month. If one disregards all the fetishes which surround this function, is it at all likely that games-playing can materially and permanently alter it?

A young woman doctor educated at a large girls' school when asked her opinion said that most girls played games while menstruating, but those who did not like games were glad to make their periods an excuse. Groups of athletes have been questioned about menstruation and statistical reports published. Kral and Markalous[5] in Prague (1930 Track and Field World Championship for Women) found 29 per cent of competitors claimed their best performance was attained during menstruation, 63 per cent noticed no difference, while 8 per cent were below their best.

A detailed analysis of a very restricted group of women athletes has been published by Ore Ingman[6] of Helsinki in 1952. She chose top-class athletes only, so that all the women were of excellent physique and good health, and were subjected to the maximum physical stress in a number of different sports. Of 107 women, nine were swimmers, thirteen gymnasts, twenty-eight basket ball players, fourteen skiers and skaters and forty-three track athletes. The average age was 23·9 years and onset of puberty 14·06 years (nearly a year younger than the Finnish average).

The women had taken up serious competitive sport after puberty. Thirty-one reported changes in their menstrual cycle since adopting an athletic career. Fourteen noticed a diminution or disappearance of dysmenorrhoea and eighteen reported disturbance of menstruation during training or competition. The abnormalities were more marked during competition, which stresses the influence of mental anxiety. Irregularity, menorrhagia and dysmenorrhoea were the commonest symptoms. One hundred and four out of the 107 had participated in sports events during menstruation, seventy-seven without ill effects. No permanent changes were reported by those in whom competition produced a disturbance of the cycle. Swimming and the pentathlon affected menstruation in about one third of the participants, and Ingman attributes this to temperature changes and prolonged repeated exertion. In the Olympic Games at Melbourne six gold medals were won by women who were menstruating.

It is interesting to know how seldom does competitive sport induce amenorrhoea, although, fear, anxiety and worry are well known causes of this upset. Athletes are clearly a well-adjusted group of women besides being healthy and well-nourished.

A woman should never assume that a disturbance of her menstrual cycle is the result of athletic activity. If the menstrual change is severe or repeated she should seek medical advice, as she almost certainly will; for games players of both sexes are very symptom conscious, being anxious lest anything should go wrong with their bodies. Quite a number of competitors in top grade women's events are in the mid-thirties, are married and have children. These women are subject to the gynaecological ailments of their age group; and menorrhagia might well be due to fibromyomata rather than to running or jumping. There are many coloured athletes and fibromyomata are especially common and appear early in negresses. Provided the pelvic organs are normal, minor menstrual irregularities are unlikely to be made worse by competitive sport.

Internal sanitary protection will be worn by most athletes during training and competition, for convenience and for full freedom of movement. During vigorous exercise there must be some friction between the internal absorbent and the vagina, the anterior and posterior walls of which are normally in contact when a woman is standing. Internal tamponade may lead to vaginitis and is most undesirable when a vaginal discharge is present, since it dams up the flow and increases irritation. Athletes should revert to external protection after training, taking care that the tampon is removed complete. Fragments may become detached from the tampon during strenuous movement and may easily be retained, soon giving rise to an unpleasant discharge.

A warning should now be given against measures calculated to stop an inconvenient menstrual period due to start during a sports meeting. It has been shown how little menstruation affects performance so that there is little excuse for taking hormonal preparations in order to delay or precipitate menstruation. The result may be that a girl menstruating normally and regularly has her cycle upset for several months after the attempt to interrupt it by hormones.

PREGNANCY AND PARTURITION

Women continue to ride and play games during the early months of pregnancy without mishap. At Melbourne three competitors were pregnant. It would be wise for any woman knowing herself to be pregnant to refrain from any sport involving bumps and falls for fear of miscarriage. As pregnancy advances, a woman finds movement more effort and has less desire to play games. Toxaemia of pregnancy does not occur more frequently among athletes, but violent exertion during the later months of pregnancy is unwise because it may cause a rise in blood pressure.

Pregnant athletes show a marked absence of worry and tenseness during pregnancy. They enjoy doing pre-natal exercises and attending relaxation classes and they naturally carry out this preparation far better than non-athletic women. Athletes have developed control of their muscles and they appreciate the difference between contraction and relaxation. That the athlete usually has a narrow pelvis is untrue. Athletic women tend to be bigger than the average; and nature seldom makes a big girl with a small pelvis. The hips may look narrower because superfluous fat is shed in training; but in obstetrics it is the capacity of the bony pelvis which counts, not the upholstery.

An important paper on 'the course of delivery of Finnish baseball players and swimmers' has been written by Kalesi Nieminera[7], who examined the obstetric histories of fifty-three baseball players and forty-one first-class swimmers. The average age at marriage was twenty-four years—much the same as the average for the general population. The average age at delivery of the first child was twenty-six, which proves the relative fertility of these athletes. The incidence of toxaemia of pregnancy was not raised. In Finland there is a high incidence of narrow pelves: Nieminera noted the external pelvic dimensions of the athletes and found them to be above the average. External measurements are only a general guide and have been discontinued in England. The intertuberous diameter in these women is not recorded and would have been a guide as to any narrowing of the outlet. However, the subsequent course of labour showed most of them to have an adequate outlet.

Duration of labour was four hours less than the normal. The second stage averaged fifty-five minutes in the baseball players and only forty minutes in the swimmers; which is a very good performance for any group of women in any land.

Perineal relaxation was particularly favourable. Forty-one had no tear and only seventeen episiotomies were made. Three caesarean sections and three low forceps deliveries were performed. The incidence of caesarean section, forceps application and eipisiotomy is not a reliable guide to obstetrical efficiency as practice varies so much and indications for interference are so differently interpreted by obstetricians; but normal unaided delivery was the rule in this selected group of athletic primiparae. The third stage was normal, the babies were of average size and only four were premature.

This report dispels the fable of the athlete's narrow pelvis and rigid perineum. Equanimity of mind, controlled breathing, strong abdominal muscles, the ability consciously to relax antagonistic muscles of the pelvic floor and to dispense with purposeless contraction of the limb muscles render the athlete an ideal parturient. It must be many years since medical men would have thought otherwise, but many of the public still believe that an excess of sport in youth is bad for child-bearing.

While most women who have children consider their athletic career over because the cares of household and children prevent extensive training, let us remember that Mrs Blankers Kohn had two children before winning three Olympic gold medals in London. Those who wish to continue sport after childbirth should allow at least three months to elapse before starting serious training. They will all have performed post-natal exercises from a few days after delivery. It should be unnecessary to add that sporting activity must be restrained during lactation. A perineal laceration or episiotomy that has been well repaired and is soundly healed will not constitute a bar to further sport.

In many girls the desire for games is not lost after a confinement—they constantly ask when they can start to swim or to play tennis or golf again. Their impatience should be curbed for three months and a pelvic examination made before they are allowed to resume strenuous games.

Not all athletes will escape with an uncomplicated pregnancy and confinement. Severe toxaemia and accidental ante-partum haemorrhage would make further indulgence in competitive sport unwise. Such sequelae of parturition as uterine malposition or erosion of the cervix or haemorrhoids should be treated before training begins.

REFERENCES

1. Ernst Jokl: The Athletic Status of Women. *British Journal of Physical Medicine*, November 1957.
2. Report of American Amateur Athletic Union, 1953.
3. Gunnar Malmstrom: Sport. *Medicine*, Helsinki, 1953.
4. The Physical Education of Girls, 1922. *British Medical Journal*, 2, 321.
5. J. Kral and E. Markalous: Der Einfluss de Menstruation auf die sportliche Leistung. *2nd International Congress Medicine Sport Proc.*, Leipzig, 1937.
6. Ore Ingman: Menstruation in Finnish Sportswomen. Sport. *Medicine*, Helsinki, 1953.
7. Kalesi Nieminera: Course of Delivery of Finnish Baseball Players and Swimmers. Sport. *Medicine*, Helsinki, 1953.

8

NUTRITION AND THE ATHLETE

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SELDOM has Science gained ground so slowly from superstition as in the field of the effects of diet on athletic performance. Ancient shibboleths of trainer and athlete alike, their origins deeply rooted in folklore, persist despite progressive enlightenment in many other spheres of human activity. Interest in this subject was taken by the Greeks several hundred years before their Olympic games ceased to take place, when certain athletes advocated large quantities of meat as an aid to athletic achievement. In the mid nineteenth century A.D. this theory was again propounded by writers on the subject together with such other admonitions as purgation and the omission of milk. Since that time the dietary predilections of victors have been imitated by other competitors seeking similar laurels and the belief in some secret elixir of prowess has been fostered by the specious advertisements of commercial interests. Vitamins too have had their vogue and continue spuriously to do so; the Oxford Boat Race crew during their eight successive victories from 1861, were trained on a diet devoid of vegetables and fruit—'So much for vitamins in athletics!' say Drummond and Wilbraham.

Although impairment of athletic performance must inevitably result from impaired nutrition, it is not a corollary that over nutrition leads to improved performance. In any case the absolute limit of performance is dependent on factors other than those purely of nutrition, of which the most important are probably psychological. Let it be said at the outset then, that the principles governing nutrition in athletes are those governing the human body as a whole but adapted to meet the special stresses and strains to which athletes are subjected. Within the confines of these principles, however, allowance must be made for the fact that an athlete is a highly individual person and that his personal factor must be taken into consideration when determining his food intake both during training and at the time of contest.

THE PERSONAL FACTOR

Many an athlete has a higher than average sensitivity and the emotional stress to which a competitor is subjected on the day of competition, let alone motivation factors before and during training, can influence considerably both appetite and gastric function. His personal characteristics must be catered for and his likes and dislikes humoured within reason. Furthermore the determination of his nutritional requirements must be influenced by the difference in physiological demands imposed by different kinds of exertion; energy demands for a single effort event such as hammer throwing are dissimilar from those of marathon events whether on the track or in such cases as long distance bicycling.

The adaptation of nutritional principles to specialised athletic performance must, therefore, depend on personal psychological factors, on the quality of food eaten, its quantity and the times at which it is consumed. In regard to the first of these points, appetite is usually a safe guide and where preference in the efficacy of a specific food or so-called dietary supplement is concerned this may be indulged providing that basic principles, which will be discussed shortly, are not transgressed. The quantity of food, with a similar proviso, should also depend on individual appetite, since there is considerable variance in individual requirements, some human machines being able to work at a more economic rate than others. At the beginning of training the nutritional status of an athlete, in relation with the type of event for which he is being trained, must be borne in mind in advising him about his diet. The imposition of a stereotyped diet, dictated by the trainer's fancy, does not meet the case at all.

Specialised techniques exist for assessing the nutritional state of specialised athletes and can be employed at the beginning of the season so that a suitable regime providing appropriate energy can be prepared. In this connection it can be said that the most suitable guide in assessment is not the standard height-weight-age table but the individual characteristics of the body concerned. In the broader field of athletics as a whole it now becomes necessary to delineate basic principles and terminology.

SIMPLE PRINCIPLES OF NUTRITION

Food is the fuel on which the human machine runs. After being eaten, its basic constituents protein, fat and carbohydrate (their proportions varying with each foodstuff) are transmuted, by the process of digestion, into substances which can be absorbed and utilised by the body tissues. The bio-

chemical process by which food is changed into assimilable nutrients is undertaken with the liberation of heat and energy, the units of which are measured by calories. About 25 per cent of the energy contained in food can be converted into mechanical work. Using the calorie as a term of measurement, it is known that 1 gram of carbohydrate and 1 gram of protein yield 4 calories each and that 1 gram of fat yields 9 calories. Thus if the composition of a particular food is known, it is possible to calculate the number of calories derived from it. The amount of energy expended governs the amount of food required and energy is expended even when the body is lying at complete rest, not only when exercising. If more food is eaten than is required for the individual's activity it will be converted into fat and stored as such in the body. Conversely if less food is eaten than the amount of energy expended, loss of weight ensues because the stores of body fat are used up. Hence the degree of physical activity of an individual athlete is the yardstick by which his overall calorie requirement and food intake must be measured.

Protein

Proteins are complicated organic compounds composed of a union of amino-acids. These are essential to life since the body cannot manufacture them for itself and they must be supplied in the diet. Not all these amino-acids exist in the same food, some preponderate in food of animal origin and some of cereal. A combination of both animal and cereal protein is necessary to ensure total optimum effect. Protein is needed by the body for the growth and repair of body tissues and its value in providing energy is less than that of carbohydrate and fat. Foods which are high in protein are meat (including fish and fowl), cheese, milk, eggs, peanuts. Significant quantities are contained in wheat flour.

Carbohydrates

Carbohydrates mainly comprise starches and sugars and provide the cheapest and most convenient sources of fuel for energy purposes. Examples of high carbohydrate foods are sugar (which includes sucrose or cane sugar, glucose and fructose), flour and all its products including bread, other cereals, dried fruit and potatoes.

Fats

These, like carbohydrates, contain carbon, hydrogen, and oxygen, but with relatively less oxygen in relation to the amount of hydrogen. The difference between one fat and another is due to the different fatty acids present. Predominantly fatty foods are butter, margarine, cream, lard and oils.

Minerals and Vitamins

Some twenty inorganic mineral elements (such as calcium and iron) all of which must be derived from food, are required for the proper functioning of body processes. Salt (sodium chloride) is an important example since it is lost from the body in the urine and whenever sweating occurs. Lack of it may result in dehydration and cramps. Vitamins are organic substances present in food in small quantities which do not provide calories when eaten but which are necessary for life, for the efficiency of certain body functions and for protection against specific diseases. Vitamins of the B group play an important role in the energy releasing reactions associated with the utilisation of carbohydrate and fat. An excess quantity, however, does not speed up these processes.

For proper health and efficiency an individual must have all the nutrients mentioned above in amounts sufficient to satisfy both specific needs for them and calorie requirements, in proportion one to the other so that 50-55 per cent of the total calories are derived from carbohydrates, about 35 per cent from fat, the remainder from protein. For adults leading an ordinary life it is estimated that about 1 gram of protein per kilogram of body weight is required. The special requirements of athletes are considered below.

FEEDING IN RELATION TO THE TRAINING PERIOD

Considering the application of these basic principles to the athlete we are first faced with the fact that his overall calorie requirements vary from 3,000-6,000 daily depending on the intensity and duration of his daily exercise. The average expenditure of calories in playing tennis is about seven per minute, of cross country running about eleven, and of swimming (the crawl for fifty-five yards) fourteen. An appropriate amount of food must, therefore, be taken daily to provide for this expenditure of energy, in addition to his basic requirements. Intake must also be governed to some extent by the size of his body since the expenditure of energy in moving it is proportional to its weight.

About 12-15 per cent of calorie needs should come from protein sources, one-third of which should be of animal origin. This is slightly higher than the average diet since, as the period of training progresses an increase in muscle bulk will occur and thus an increased demand for protein to maintain it. It should not be inferred, however, that eating large quantities of steak will 'make muscle' nor that it will improve performance. On the other hand protein intake above requirements is not likely to be harmful provided that the increase does not cause the diet as a whole to become unbalanced.

A high calorie diet is necessarily somewhat bulky unless fats are increased. Nowadays the danger of a high fat diet is much in the news but whereas a comparatively high fat intake may have serious disadvantages to those who are habitually sedentary this is not the case with athletes in active training.

Meals should be taken at regular hours, three times daily, though if the sport for which training is undertaken is of long duration it may be wiser to have lighter meals at more frequent intervals.

A regular daily intake of salt is required to offset normal losses in the urine and additional losses in sweating caused by exertion. This can normally be met by adequate seasoning at meal times but in a hot climate it may prove necessary to take an extra ration in the form of a daily salt tablet.

Liberal amounts of fluid are beneficial and the custom of some trainers in restricting it is to be deprecated. As much as $1\frac{1}{2}$ pints of fluid may be lost through sweating and if this is ignored dehydration may ensue—even small degrees of which lead to impaired efficiency. While on the subject of fluids it is appropriate to mention alcohol. The core of this matter is usually veiled with prejudice and bigotry. The fact is that small amounts of beer, wine or diluted spirits do not interfere with training and for those who like and are accustomed to taking them, they may be beneficial from a psychological point of view. It must be understood, however, that alcohol plays no part in improving physical efficiency and may well seriously affect actual performance through its adverse effect on co-ordination.

Some individuals, including athletes, respond to stress by over eating. If, for this reason or because of greed, food intake exceeds energy expenditure, there is a gain in weight and accumulation of fat. However, the fact that an athlete is overweight according to the standard tables of averages is not evidence of obesity since muscle bulk and bony framework must be considered. When it is clear, especially at the beginning of training, that an individual is overweight because of fat then it is essential that this be reduced. The only reliable and safe method of achieving this is by reducing carbohydrate intake and this should be set in hand as early in the season as possible. Sugar, jam, honey, potatoes, bread, cakes, biscuits and other cereal foods should be restricted to a minium until the desired weight is attained. A diet devoid of ordinary cereal foods tends to become unacceptable but these may be usefully substituted by Energen Starch-reduced products. Crash diets, food substitute powders, pills, dehydration and other stunts should be avoided like the plague. Apart from health hazards one must deplore the use of such methods in an attempt to reduce weight below normal by individuals seeking to gain competitive advantage over those in a lower weight bracket.

So far as overall feeding is concerned, the best way to secure an adequate

diet is to eat as wide a variety of foods as possible. If good helpings (amounts varying with personal requirements and response to training) are taken daily from each of the following food groups, the aspirant to championship cannot go far wrong from a nutritional point of view.

1. Milk and cheese
2. Meat, fish, fowl, offal and eggs
3. Fruit and vegetables
4. Butter or margarine
5. Bread, other cereal foods and sugars.

DIET IMMEDIATELY PRIOR TO CONTEST

The relative compositon or size of a meal taken prior to intense activity of short duration is unimportant. But for those events which require prolonged activity and considerable endurance, a high carbohydrate diet maintains performance best, provided that such a diet has been consumed consistently for several days before the event. In other respects choice of food at the final meal should be influenced by experience gained during the training period. Highly concentrated foods, however, are best avoided.

Strenuous exercise immediately following a meal alters the manner in which the stomach's digestive processes work—doubtless due to competition between it and the muscles for an adequate blood supply. This and the consumption of injudicious foods may in turn produce abdominal distension with consequent embarrassment of heart and lungs by upward pressure on the diaphragm. Hence the last meal should be taken three to four hours before an event. Sugar, glucose and similar supplements are of no value before events of short duration or where the output of energy is modest.

In marathon events and where extremes of energy output are sustained for long periods, the final meal should have a preponderance of carbohydrate. Further, the inclusion of bouillon (chicken broth for example) at this meal helps in maintaining salt balance. Excess protein is to be avoided owing to demands made on the kidneys in excreting some of the products of its metabolism. Salads and highly spiced foods, with their effect on the bowel, are also best avoided for forty-eight hours before contest.

Sugar supplements before and during events involving prolonged efforts are extremely useful since carbohydrate reserves in the body are a limiting factor. Honey has no advantage over sugar in this respect and dollops of honey, lumps of sugar or pieces of glucose require fluid for their assimilation; they may thus invite some dehydration (albeit minimal) by drawing fluid to the stomach from other tissues. A solution of glucose is, therefore, the most

appropriate form of ingestion. During the course of marathon contests drinking too highly concentrated solutions may cause nausea and therefore it may be best to drink diluted fluids for example sweetened weak tea with lemon taken frequently at intervals. Recently a 'Pregame Meal' has been developed in America to avoid vomiting in athletes before competitive events. This is a liquid meal supplying 925 calories which passes through the stomach in less than two hours. Although possibly of benefit to a few individual athletes, such a meal is not likely to be generally acceptable. Caffein-containing drinks do not improve physical performance since their 'stimulating' effect is purely that of decreasing awareness of fatigue. Alcohol before an event must be forbidden.

Finally it may be said that, within the limitations of the foregoing principles, acceptability of any diet and the personal psychological factors associated therewith are of prime importance to the individual athlete.

SUMMARY

1. The principles of nutrition in athletes are the same as those of ordinary human nutrition but they may be adapted to meet the needs of exceptional physical stress.

2. The personal factor in choice of food and of dietary management within these principles is important.

3. Although malnutrition (including obesity) impairs efficiency, the supplementing of an adequate diet by additional food or stunt preparations does not improve performance.

4. Sound feeding to attain peak efficiency must be undertaken *during the training period.*

5. Certain factors in regard to the last meal eaten before an event affect performance and vary with the type of event involved.

REFERENCES

Boje, O., *League Nat. Bull.*, Health Org. 8, 439, 1939.
Davidson, S., Meiklejohn, A. P. and Passmore, R., *Human Nutrition and Dietetics*, 1959.
Drummond, J. C. and Wilbraham, A., *The Englishman's Food*, 1939.
(Editorial) *A.M.A. News*, 2.2, 1959.
Mayer, J. and Bullen, B., *Physiol. Rev.* 40, 369, 1960.
Passmore, R. and Durnin, J. V., *Physiol Rev.*, 35, 801, 1955.
Rose, K. D., *et al.*, *J.A.M.A.*, 178, 30, 1961.
Yudkin, J., *This Slimming Business*, 1958.

PART II

1

MECHANISM OF PRODUCTION OF INJURY

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OUTDOOR sports in every form involve some risk of accidental injury but the same may be said of every form of human activity. It might be supposed, for example, that one was safest indoors at home and in most danger travelling on our overcrowded roads or engaging in some dangerous occupation. In fact, of the forty-five fatal accidents which occur each day in England twenty-four happen in the home, sixteen while travelling and only five at work. A fatal injury rarely occurs in the course of athletic activities and there are far more simple accidents in the home than on the playing fields.

Obviously different games and sports involve different degrees of risk, and in the following sections experts give their views on the dangers in their own particular field. While the editors do not necessarily agree entirely with all that they say their opinions carry the weight of experience.

All sporting injuries have, however, certain features in common and the mechanism of production is important from the point of view both of prevention and treatment.

MECHANISM OF PRODUCTION OF INJURIES

There are three basic ways in which injuries may be produced—they may be caused by an outside agency, by body activity itself or by long maintained mechanical stresses.

Outside agency. This is much the simplest and most usual cause of injury and may take many forms. Essentially the damage is due to the influence of somebody or something unconnected with the person injured and usually not in any way under his control. The outside agency itself may be the sole cause but body activity, body momentum or both may also be involved. For example, a fielder standing motionless near the wicket may be watching the batsman running and be struck and injured by a ball thrown from the outfield. In this case the outside agency, the cricket ball, is solely responsible for his injury. If, on the other hand, a goalkeeper, in attempting to punch a foot-

ball away miscalculated and punched the goal-post, injuring his hand, the effect of the outside agency, the post, would be associated with the body movement, that is the act of punching; and the injury produced would be the result of these two things. Finally, if while jumping, a rider was thrown from his horse, the injuries produced in the fall would be due to the effects of the ground, which is the outside agency, combined with body momentum. In many circumstances momentum and body activity might be combined to produce injury in conjunction with an outside agency.

The injuries produced by an outside agency can be classified in many ways. For example, they may be direct, when the damage is done at the point of impact; or indirect, when the damage occurs some distance away from the point of impact, force being transmitted through the tissues themselves. The injuries may be 'high velocity' or 'low velocity' depending on the speed at which the outside agency and the body come into contact; and this factor determines to some extent the nature of the tissue damage.

The danger of injury from an outside agency varies tremendously in different games and sports. Quite often such injuries are produced by contact with another player and are (or should be) quite accidental. Although accidental, such injuries are unavoidable. In Association Football for example it would be impossible to prevent accidental contact between the players occurring frequently during the course of the game. In other sports the danger of this type of injury is sometimes used as a tactical weapon: when a fast bowler bowls a bumper he does not do so with the idea of hitting the wicket. In other sports the danger is almost negligible; for example injury due to an outside agency must be very uncommon in swimming races.

Self-produced injuries. Injuries of various kinds may occur simply as the result of body activity, without the intervention of any outside agency at all. The basic force producing such injuries is muscular contraction, and this may cause damage in some circumstances without being itself in any way abnormal. A powerful contraction of the peroneus brevis, for example, may avulse the base of the fifth metatarsal. Much more commonly, however, they are due to some unbalanced or abnormal body movement. A fast bowler may miss his footing during delivery. His action then becomes unbalanced, and he may tear a muscle in the abdomen or in the thigh. Finally, such injuries may be caused by some action which, while not unbalanced, is abnormal and, therefore, throws too much strain on the body tissues. For example, the sudden movement of the low back requiring extreme rotation and lateral flexion which is necessary when the so-called 'shut-face' technique is employed in striking a golf ball very often causes various low back disorders of traumatic origin.

FIG. 1. All the factors which may be responsible for accidental injury may arise together. Any one of the players in this situation might sustain an injury which would be completely accidental.

Long continued stresses. Injuries due to an outside agency or body action are comparatively simple: the body tissues are subjected to a mechanical force which is greater than their inherent strength and they are thereby damaged.

Long maintained stresses are, however, much more subtle in their effects. The only tissue in the body which can sustain stresses indefinitely without being harmed is bone. Articular cartilage and the intervertebral discs also have to sustain such stresses and are highly modified for this purpose, although they often suffer ill effects from so doing. All the other soft tissues of the body, muscles, tendons, ligaments, and so on are harmed if they are subjected to mechanical forces for too long. Such a force is usually one of traction and may be comparatively small. If applied for a short time it does no harm: but if it is continued indefinitely it sets up in the affected tissues a cycle of changes which leads to pain and disability. The conditions so produced are usually called strains and are discussed in a later section of the book. They are, however, basically traumatic in origin, that is to say they are results of mechanical force applied to the body tissues.

Once injuries have occured, treatment is, of course, important. It is, however, equally important to prevent them, since sport should bring pleasure not pain. Many of the injuries due to an outside agency may be prevented, or their effects at least minimised, by the use of protective padding or protective clothing; and this is common practice in many games, the extreme example being American Football. Injuries can also be minimised by the general outlook of the players: the importance of a sporting approach needs no emphasis. They can be controlled to some extent by legislation; practices which are inherently dangerous can be outlawed. The prevention of self-produced injuries is largely a matter of technique and training. Most effective actions are natural and unlikely to cause damage; and any technique which involves unbalanced or convulsive movement is to be avoided. Finally, the prevention of the chronic stress injuries is up to the individual, being a matter of training in posture and manner of movement.

2

ASSOCIATION FOOTBALL

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THE INJURIES common in football are of four main types:—

1. muscular injuries. These may be strains and tears produced by sudden fast movements, or contusion of muscle with haematoma formation as a result of body contact.
2. joint injuries involving cartilage capsule and ligaments.
3. bone injuries, whether fractures or haematomata.
4. concussion.

MUSCLE INJURIES

Muscle starts and ends as tendon: that is to say both its origin from bone and its insertion into bone are tendinous. It may be injured at either end or at the fleshy part, called the belly. The muscles most often injured in football are the hamstrings, the quadriceps group and the calf muscles.

Complete rupture of a muscle is rare, except of the achilles tendon, which may be torn by a sudden movement or by direct violence. Very occasionally a footballer may rupture the quadriceps. Whenever the fleshy part of a muscle is torn, partly or completely, there is a good deal of bleeding. The resulting haematoma needs immediate treatment. A disabling complication of a torn muscle is myositis ossificans—the formation of bone in the muscle tissue; and it is important to watch for the first signs of ossification, which can be seen in an X-ray photograph. Some muscles are especially liable to develop myositis ossificans—the adductor longus tendon, for example, which may be damaged when a player over-reaches for the ball; or when he makes a tackle, particulary a sliding tackle.

It is not easy to prevent muscle damage: but proper graduated training makes the muscles less liable to fail when strain is put upon them. With training they become more supple and more elastic—more tolerant, as it were, of unusual loads. Some players seem far more prone to injury than

others—especially the short hamstring type, who have well-developed thigh muscles but are relatively short from hip to knee. Players of this build ought to concentrate on stretching exercises.

JOINT INJURIES

The ankle and knee joints are the ones most often injured, whether by direct or indirect violence. The agent of direct injury is the outstretched foot in blocking a shot or tackling: the mechanism of indirect injury is a rotation, as when the player pivots quickly.

Knee joints injuries are of three main types:

a. acute flexion injuries which sprain the joint.

b. abduction injuries which sprain the medial ligament. This is the commonest injury.

c. rotation injuries with displacement or tearing of a meniscus, combined with tearing of the medial ligament.

The sprain of the medial ligament is usually a combination of direct and indirect violence. It results from a blow on the outside of the knee as the player's leg is stretched out, the blow being caused either by a tackle or a falling opponent. The torn meniscus is usually the semilunar, the one on the inside of the joint: it is torn by rotation of the femur on a bent knee, as when a full-back tackles and pivots at the same time. If the meniscus is only nipped, it may slip back into place and cause no further trouble: but it may have to be manipulated. If the front end is nipped and crushed, there is likely to be limitation of the last few degrees of extension; and unless it is successfuly reduced the joint may swell. Several months' treatment may be necessary before the joint recovers completely: sometimes conservative treatment fails and operation is the only certain cure. A meniscus which has been nipped and locked several times may loosen and the quadriceps may become lax. It is important to restore the function of the muscle, on which the knee greatly depends for stability, by daily faradism and remedial exercises.

The ankle is particularly vulnerable in football players. Fractures into the joint, or just above the joint, are often caused by sliding tackles or by the deliberate act of 'playing over the ball': sprains are caused by forced abduction or adduction, movements which may result in ligament sprain, joint sprain or cracked or chipped malleoli. The anterior fibres of the lateral collateral ligament seem to be injured more than any other, and the ankle becomes tender and swollen over the upper attachment of the ligament to the external malleolus. The posterior and middle fibres may also be

involved, as a result of direct violence when the opposing player's boot comes through in a tackle from behind, the toe of his boot meeting the ankle. If these injuries are not expertly treated the player may be left with a stiff ankle. Goalkeepers who dive at the feet of opposing players or to save a shot often dislocate a shoulder.

BONE INJURIES

Apart from an occasional freak accident, the broken leg is the worst injury encountered in association football, whether it occurs as a result of sliding tackles, playing 'over the ball', or a fall over an outstretched leg.

There are a few simple measures which help to prevent injuries to bone: strong shin guards, crepe bandage round the ankle, and, for the goalkeeper, shoulder pads to absorb the violence of direct blows.

CONCUSSION

When players jump to head the ball, they may collide and knock their heads together. A ball, weighted by mud may be heavy enough to cause concussion if it is inexpertly headed, that is to say, if a player allows it to hit him instead of actively heading it. Goalkeepers, trying to sweep the ball from the foot of a man about to shoot, may be kicked on the head.

ASSOCIATION FOOTBALL

JIMMY HILL

Chairman of the Professional Footballers' Association
Qualified Footballers' Association Coach

JIMMY HILL'S face and beard have become widely known, not only on the football field but also in the lengthy negotiations between the players' trade union and the football league clubs. As the players' spokesman, an experienced inside forward and as a staff coach for the Football Association, he seems well qualified to give the players' views upon the injuries common in soccer.

Direct injuries, by contact with boot or ball are not, on the whole, often received, nor are they very serious when they do occur. Soccer professionals measure the seriousness of an injury by the time it keeps them out of football; and, by this reckoning, direct injuries often seem trivial and may be neglected.

The shins, which are the most vulnerable to kicks, are well padded, so that the commonest direct injury is a severe bruise of the foot: and this bruise may be 're-activated', as it were, in each subsequent match. At the beginning of the season, when the grounds are hard, and during frosty periods, a player who falls may bruise his wrist, fingers or elbow.

FIG. 1. Association football inevitably involves some risk of injury to the players.

The most spectacular accident in soccer is the collision of two players, both jumping to head the ball. The force of the contact may split the skin above or below the eye, an injury similar in production and result to that seen in the boxing ring. The nose may also be damaged in an incident of this sort.

Indirect injuries account for the greater part of enforced absence from the team. Pulled muscles—hamstrings, calf, groin or back muscles which give a great deal of trouble, are to be expected early in the season, particularly if a player has not 'worked up' gradually, if he is not fully fit or if he is fatigued. Soccer players do not, as a general rule, warm up before a game as runners do; but they try to pace themselves by a gentle start, slowly increasing the tempo during the first quarter of an hour. Muscle injury is not uncommon just after half-time, if the player makes a maximum effort before his muscles are ready for it after their short rest. The neck muscles sometimes become stiff and painful, whether as a reaction to vigorous 'heading', or because there may be periods on a cold windy day when a player, who is sweating hard, quickly cools off as the game swings for a time in another direction.

Jimmy Hill has found from his own experience of muscle injury that it is important to learn to relax a muscle recovering from damage. There is a natural tendency for such a muscle to go into protective contraction when it might easily be injured again by a sudden violent movement.

Muscle damage, however, is seldom very serious in the long term: it is the joint injury, of knee or ankle, produced by a twisting movement which is most likely to put a player off the field and keep him out of the game for many weeks. Ligament and cartilage injuries in the knee, ligament and bone injuries in the ankle—these are responsible for most of the lost playing hours. Worst of all is a combination of a twist and an impact, as when a player, turning his body, his boot studs gripping the ground, is hit upon the knee or ankle. This combination of direct and indirect injury may so disorganise the ankle or knee joint that the player is incapacitated for many months: he may never play effectively again.

3

INJURIES TO ATHLETES

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THE COMMONEST injuries on the running track are:—

1. 'pulled muscle'
2. 'teno-synovitis' of the Achilles tendon.

Injuries do occur in the field events, but there is no general pattern to be discerned save in the case of the long-jumper who comes into the category of sprinters.

1. '*Pulled Muscles*'

This term is applied to rupture of muscle fibres of the quadriceps high up in the thigh, the hamstrings, or the calf muscles; it occurs generally in sprinters during extreme effort, and to a lesser extent in runners at longer distances. The extent of the injury varies from a few damaged fibres to a very considerable tear demanding surgical intervention with suture of the severed ends.

The cause is probably an imbalance between the protagonist and the antagonist group of muscles, that is to say, failure of one group to relax when the other group contracts. In this way rupture may occur at any situation between muscle origin and insertion.

Prevention. There is a variety of opinions upon the mechanism of the injury and a corresponding variety in methods designed to afford prevention.

A favourite measure is graduated activity at the beginning of an athletic season to educate the muscles for the strain to which they are to be subjected. This precaution, if of doubtful advantage, is at least sensible. Another is sufficient 'warming up' before a contest or indeed before exertion of any kind. An objection to the explanation of injury implicit in this measure is that 'pulled muscles' are no less infrequent in hot than in cold weather. Again one can say that this, if no more, is a reasonable precaution.

Special exercises to strengthen the muscles concerned are today the fashion.

FIG. 1. The poised fully-trained athlete is not very likely to be injured.

I have had no experience of 'muscle building' and so perhaps I am hardly entitled to express doubts whether this procedure has any advantage.

Although my opinion is not universally accepted, I believe that sepsis is a responsible contributory factor. The teeth, the sinuses, and the organs of digestion should always receive attention. Of course it may be objected that to the toxin-crank there is hardly anybody in whom some focus cannot be identified if a sufficiently exacting search is made; but I take my stand on the recollection of having found a very obvious site of sepsis in instances when a breakdown of this kind has occurred.

It is sometimes alleged that special articles of diet and certain vitamins influence repair to a degree that might be regarded as prevention but I cannot persuade myself of the validity of such an association.

If one accepts the principle of imbalance of action, it hardly seems possible to adjust a runner's action to ensure better co-ordination. The nearest approach to prevention is to inhibit a recurrence by an elastic support or strapping of the site of any injury.

2. *Teno-synovitis of the Achilles Tendon*

Whilst complete rupture of this tendon may occur in older subjects in whom there is some degenerative or pathological change, this explanation is most unlikely in the case of an athlete in whom the lesion that occurs is rupture of a few fibres or inflammation of the bursa between the tendon and the bone.

The method of production is obvious. The powerful plantar flexors the calf muscles, are the main driving force in locomotion, and the tendo-Achilles is necessarily subjected to constant stress and friction through the action of running on the toes, although the heel comes into contact with the ground more than one realises either subjectively or from observation. It can be plainly demonstrated by photography.

Prevention. I cannot see how such a lesion may be prevented short of refraining from running or by attempting to ensure a flat foot, advice rather reminiscent of the prevention of seasickness by refraining from going to sea. A running shoe (or pump) must be heel-less, so a mechanical support is lacking and cannot be supplied. But I would add that the observations on general health and the influence of sepsis apply to this as to all lesions.

ATHLETICS

JIM PETERS

British Marathon Record Holder
Former Holder of World's Best Marathon Time
Empire Games, Vancouver 1954
Olympic Games, Helsinki 1952

THE THREE most common injuries in athletics are pulled muscles, strained tendons and stiffness.

This stiffness, or shin soreness as it is known, may not sound very serious; and it is true that physically, the other two injuries are more damaging. But there is a mental factor which makes stiffness a very real disability for the distance man. When every muscle feels like brittle cardboard one does not need much persuading to forgo a ten-miles training run. In this way many runners have been lost to the sport.

I know so well that 'morning-after-the-race' feeling when I was so stiff that I could not get up to make a cup of tea. Even today, when I sit back in an armchair and flex my toes, a sharp pain comes into my instep and shoots up my leg.

Many distance runners often suffer from strain of the Achilles tendon: but it is the sprinters who are most vulnerable to the third common injury, the pulled muscle. This is the result of upsetting the delicate balance of the push and pull action of the muscles. The hamstrings are often the group damaged being sometimes ruptured especially when they are at full stretch.

THE CAUSES OF INJURIES

Pain is associated with injury in two ways: first it comes as a warning that the capacity of a structure has been pushed to the limit: then it occurs when that limit has been exceeded.

Shin soreness is in the first or warning category. I suppose that it is not natural for man, or civilised man at least, to engage in the intensive and extensive exercise that modern athletics demand, particularly of distance runners. I used to run in training about 130 miles a week—the equivalent of over a million running strides a year, most of them on the unyielding surfaces of roads. Excessive running can also bring on tendon trouble: the 'shock

absorber' between the heel and the foot loses some of its spring under constant pressure and sometimes gives out altogether—much like the shock absorber of a car which has been driven over long distances or over very rough ground.

In my opinion, the chief cause of pulled muscles is cold weather. Unless the muscles are working smoothly and in harmony there is the danger of friction between them: and this is far more likely to happen in the cold weather when joints and muscles stiffen up.

PREVENTION OF INJURIES

As cold weather causes most pulled muscles, the obvious preventive measure is to keep warm. This is not so easy in Britain where the generally cool conditions are not only unfavourable to muscle movement but, as a result discourage the fastest times. The only thing the runner can do is to cut to the minimum the risk of damaging cold muscles. In training he should wear a track suit whenever he feels the slightest doubt about the conditions: and he should keep it on until his body has been well warmed up and is working freely.

This applies equally to competition. Before the race it is important to get completely warmed right through, starting by jogging and then increasing the pace. At the finish of the race the runner should 'run down' and avoid pulling up sharply.

As far as shin soreness is concerned, I do not think there is any way of preventing it: I certainly never found one. It can be alleviated, by hot bath or massage, but apart from this there is nothing to do but grit ones teeth and battle on.

On the other hand I did come across a way to prevent tendon trouble. I cut the inner sorbo sole out of an old pair of plimsolls and put them into the ones I used for training. This took much of the strain off the tendons, the sorbo doing much of the shock absorbing. After adopting this precaution early in my marathon running career I never once suffered from tendon trouble.

This measure I recommended not only to marathon and other road runners but also to distance runners on the track. In my view many track runners do far too much training in spikes. Spikes give fine adhesion and spring, but they impose a great strain on the heel, which has virtually no support.

The three physical disabilities I have mentioned are associated with running under all circumstances. Competitive athletics however bring their own dangers, such as the effect of the sun and the chance of being spiked.

The sun, the sprinter's friend, is the distance runner's enemy, a fact that was painfully brought home to me in the Empire Games at Vancouver in 1954. Distance men need not and should not be called upon to perform in the heat of the day, as we were in Vancouver. At the 1960 Olympic Games in Rome, the marathon was staged in the evening.

Spiking occurs mainly in middle distance events, generally at the first bend when competitors, bunched together, are striving to gain a good position. It can be avoided if the field is kept to a reasonable number and if the start of the race is kept as far as possible from the first bend.

A final word: the most important tools of the athlete's trade are his feet. They should therefore be looked after with the utmost care. Many aches and pains can be prevented by making sure that socks are clean and shoes comfortable.

And blisters: brine can be used to toughen up the feet; but I always found that hard training did the job well enough.

4

BASEBALL

DR. GEORGE E. BENNETT

M.D.

THE LATE Dr George E. Bennett, the famous American Orthopaedic Surgeon who was President of the American Orthopaedic Association and American Academy of Orthopaedic Surgeons, was in his early days a semi-professional baseball player. Because of his eminence in the orthopaedic profession and his prowess and interest in this sport, he had become an authority on baseball injuries. He kindly allowed us to extract his article on Elbow and Shoulder Lesions of Baseball Players.[1]

His opinion about these injuries can be summarised as follows:—

Scapula

A condition has been described characterised by marked discomfort in the region of the infraspinatus muscle thought to be due to an irritation of the suprascapula nerve by hard throwing. The point of irritation is where the nerve curves round the base of the spinae. Relief has been gained by hydrocortisone injected into this region 1-2 cc. (50 mgms. per cc.).

Shoulder

The common conditions affecting the shoulders of ball players are:—

1. Acute and chronic bursitis
2. Supraspinatus fraying or rupture
3. Biceps tendonitis or dislocation
4. Loose bodies
5. Exostosis of the infra glenoid region.

Acute bursitis: due to trauma is successfully treated by rest and heat.

Chronic bursitis: with thickening of the bursa and symptoms of crepitus and local discomfort is resistant to treatment both local and operative.

Supraspinatus fraying: constant snubbing of the tendon against the greater tuberosity leads to fraying of the deep fibres of the tendon. The symptoms are

pain in the region of the tuberosity and the anterior shoulder. This condition again does not yield to local or operative treatment.

Supraspinatus rupture (a rare condition): with a history of a sudden snapping in the shoulder and intense pain occurring in a young person, exploration is justified, and many of these cases can be returned to good function by suture of the rupture. If a frayed tendon ruptures in an older patient the chances of returning to sport are remote in spite of suture.

Bicipital tendonitis: presents with pain in the shoulder and tenderness of the bicipital grooves aggravated by flexing the elbow against resistance. Actual crepitus may be felt, or a click if the tendon dislocates. Stapling has been successful with a dislocating tendon.

Loose bodies: these are rare, but do occur, giving rise to intermittent catching and swelling. These are best removed through an axillary approach.

Sub-glenoid exostosis: presents with a severe pain on throwing with maximal effort leading to an exostosis forming in the region of the attachment of the long head of triceps. The symptoms are local discomfort, tenderness to palpation and pain referred to the deltoid from irritation of the circumflex nerve. It can be demonstrated by an anterior-posterior X-ray taken with the patient lying on his back with the arm fully externally rotated and abducted to the right angle with the tube tilted 5° from the vertical so as to point backwards and slightly upwards. Normal use of the arm is possible, but hard throwing is not possible. Operation has not proved successful in curing this condition.

Elbow

The common baseball injuries of the elbow are:—

1. Osteochondritis
2. Olecranon fractures
3. Spur formation
4. Antecubital swelling
5. Ossicle formation in ligaments.

Osteochondritis: The commonest injury seen in the elbow of baseball players is due to repeated trauma of forced sudden extension resulting in flaking off of cartilage leading to loose bodies, cartilagenous outgrowths semi-attached, and synovial thickening. Symptoms are pain and swelling and long disability due to obstruction to full extension by cartilagenous outgrowths and loose bodies. Treatment by removal of the loose bodies or outgrowth is usually successful in restoring good function.

Olecranon fracture: the case can be either of the flake type torn off the tip

of the olecranon or the " hair line " type of fracture thought to be due to fatigue. A rare type of fracture is one involving the articular surface in a man with an abnormally long olecranon process. In the case of flake fracture causing symptoms the removal of the flake effects a cure and in the last mentioned a formal shortening of the olecranon process with attachment of the triceps resulted in a cure.

Spurs: in cases recorded these spurs are situated on the inner edge of the trochlea. They are extremely painful. They may be very small and difficult to visualise on the X-ray films. If causing trouble they may be removed. But certain large spurs cause no trouble and should be left alone.

Antecubital swelling: this unnamed condition occurs in baseball pitchers who, to gain swerve, forcibly supinate the wrist at the end of the throw. The condition can be disabling and is characterised by fulness over the pronator teres and the antecubital fossa. Exploration of the joint has revealed nothing and the suggested treatment is a division of the fascia transversely and vertically over the region of the antecubital fossa which has in many cases resulted in cure.

Ossicles: ossicles either single or multiple can form in the joint ligaments on the inner side of the ulnar nerve. They form as the result of chronic strain and probably take years to develop. They may be symptomless but if there is evidence of ulnar nerve irritation they should be removed. The ossicles are difficult to see but can be located by use of a needle to puncture the ligamentous tissue and then dissected free. The hiatus in the ligamentous tissue is left open.

REFERENCE

1. Bennett, George E. (1959) *American Journal of Surgery*. September issue Vol. 98, No. 3, pp. 484-492.

5

BASEBALL INJURIES

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THE INJURIES which occur at baseball may conveniently be considered in five separate groups.

I. *Motor Power Unit Injuries*

A number of injuries can be grouped together by extending Brewer's[1] and O'Donoghue's[2] concept of muscle-tendon unit to include muscle-tendon-bone as a true unit of motor power.

(A) *Chronic Overload* is caused by use of a muscle-tendon-bone unit beyond its endurance. Fatigue develops and results in muscle spasm, myositis, ischemia of muscles, tenosynovitis, adherent tendonitis, attrition of tendons, stress fractures, and spur formation of bones. Classic examples of this in baseball have been carefully detailed by Bennett (supra vide).

Prevention of this type of injury should be aimed at progressive conditioning, carefully balancing demands with increasing tolerance.

(B) *Acute Overload* is caused by sudden application of stress to a muscle-tendon-bone unit which exceeds its tolerance. This results in a disruption of the unit, ranging from a mild tearing of a few muscle fibres to complete disruption of the unit at its weakest point or at the point of greatest stress.

This type of injury is inevitable in any vigorous, competitive sport. Its incidence can be reduced by smoothness of function, quickness of reflexes, diligence in training, and perfection of protective equipment.

Examples of acute overload injury occurring in baseball are:—

1. 'Baseball finger'. Avulsion of the insertion of the extensor digitorum communis occurs when the ball, or other object, strikes the tip of the extended finger.

2. Rupture of the central slip of the extensor mechanism at the middle finger joint (Boutonniere deformity). This occurs either from a direct blow on the tip of the finger or from a direct blow across the joint.

3. Triceps injury—Acute strain with swelling or haemorrhage may result in myositis and frequently develops in incompletely conditioned pitchers.

Complete rupture can occur in over-ambitious and under-trained players.

4. 'Charlie horse' or acute, localized, intramuscular haemorrhage can result either from mild to moderate muscle overload or from contusion from a direct blow.

5. Anterior compartment syndrome. In vigorous early season conditioning, acute swelling, oedema and haemorrhage in the unyielding anterior crural compartment may occur and can result in necrosis of muscle and occlusion of neurovascular bundles with disastrous consequence unless relieved.

6. Plantaris tendon rupture or calcaneal tendon rupture mainly occurs with sudden spurting of speed, particularly if the athlete is incompletely conditioned or inadequately warmed up.

7. Acute arch strain frequently occurs in training and early in the season.

II. *Fractures and Joint Injuries*

1. Fractures of the tip of the distal phalanx are frequent in ill coordinated. players. This fracture occurs almost universally in catchers who must handle fast pitches that are often deflected by the batter.

2. Dislocations of distal or middle finger joints occur regularly in baseball.

3. Dislocation of thumb metacarpo-phalangeal joint. The thumb is particularly vulnerable in baseball and the metacarpo-phalangeal joint seems predilected as the site of injury.

4. Fractures of carpus, forearm, and humerus occur infrequently in baseball.

5. Fractures of the vertebrae, pelvis and ribs are virtually unknown in baseball.

6. Fractures of the femur are infrequent.

7. Knee Injuries. Ligament tears and meniscal injuries are much less frequent in baseball than in body contact sports such as football. These injuries are similar to those occurring in other sports and need no special comment here other than re-emphasis of the desirability of early repair, within the first few hours if possible. This is supremely true in athletes, who require optimal possible restitution of function.

8. Tibial fractures are uncommon.

9. Ankle injuries are second only to hand injuries in the incidence of acute trauma. Moderate to severe ankle sprains are very common and lateral or medial malleolar and bimalleolar fractures are regular occurrences. These injuries usually occur when sliding into base. A spike of the shoe inadvertantly catches in the turf or in the base; thus, the foot stops, while the body proceeds. This is most often the down foot in a lateral, foot first slide, resulting in an external rotation type injury.

These injuries are best prevented by careful training in the proper execution of slides and by skillful performance of slides. Well prepared base paths, moderately movable and slightly resilient bases, and well fitted and well maintained baseball shoes are of assistance in curtailing these injuries.

10. Foot and toe fractures have no special importance relative to baseball.

III. *Head and Maxillo-Facial Injuries*

Head trauma and maxillo-facial fractures occur in baseball, chiefly, in two ways: (1) when the batter is struck by a pitched ball or (2) when an enthusiastic fielder, chasing a fly ball, rams the fence or grandstand around the field. This first cause can be controlled by strict adherence to the rules which prohibit pitchers from throwing 'bean balls' and prevent batters from crowding the plate.

Inexorable and heavy penalties for such conduct will discourage its employment. Routine use of the protective hard helmet by batters has greatly reduced the seriousness of injuries resulting from being hit by the pitched ball. Bumping into fences can be reduced by utilizing wide cinder paths to warn fielders that they are approaching obstructions.

IV. *Soft Tissue Injuries*

1. Abrasions (Strawberries). These injuries occur in sliding and are extremely common on the hips, buttocks, and lateral thighs. Proper sliding techniques, excellent conditioning, quick reflexes and good sliding pads help reduce the incidence of these injuries.

Sterile bandaging plus Austin Moore's concept of SUELMEX (SUpport, ELevation, Massage and EXercise) will generally reduce to the negligible the consequence of these injuries.

2. Spiking Injuries. The pivot man on the double play, generally the second baseman or the short stop, is most vulnerable to this trauma, though any player attempting to make a play on a sliding base runner may be involved. Sure enforcement of the rules prohibiting intentional spiking will discourage this injury. Quick and accurate reflexes will help players avoid this injury.

V. *Injury of the Immature Elbow*

Experience is calling attention to the alarming increase of severe injury to the elbows of young pitchers in the Babe Ruth and Little League age groups of 8-12 years old. These young boys with unfused epiphyses in their elbows are causing significant displacement and in many instances permanent damage to the growing epiphyses in their pitching arm by throwing too

long and too hard. Many of these young, untrained players rely solely on the fast ball and sometimes a temporarily good pitcher will pitch five to seven innings, occasionally without adequate conditioning or even without warming up. Thus epicondylar, capitellar, or trochlear epiphyses become displaced and permanent loss of function and deformity often follow.

This disastrous injury can be prevented only by intelligent, adult control of these boys' baseball participation. The leagues have become cognizant of the possibility of this injury and will be able to diminish it by prohibiting the fast ball, limiting an individual pitcher's participation to two to four innings or a certain number of pitches and by insisting on adequate training, coaching, and conditioning.

In summary, the injuries most common in baseball have been outlined. As baseball is a fast game requiring dextrous handling of a hard, rapidly moving ball and fast running, cutting and sliding, injuries of the hands and ankles are frequent. Fortunately, more serious injuries are unusual.

REFERENCES

1. Brewer, Bruce J., Athletic Injuries; musculo-tendinous unit, Clinical Orthopaedics, 23: 30, 1962.
2. O'Donoghue, D. H., Surgical Treatment of Injuries to Ligaments of the Knee, Journal of the American Medical Association, 169: 1423, 1959.

6

BASKETBALL

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BEFORE ATTEMPTING to enumerate the various injuries encountered in basketball, it is necessary to know the forces which are applied to the various attitudes and positions assumed in this sport. The game is played with 'the fast break' technique. This calls for endless endurance, as the scene of play changes swiftly from one end of the basketball court to the other. The ball may be rapidly thrown and always the participants strive to gain possession of the ball and place it through the hoop.

In order for each member to perform his duties effectively, he must be in perfect physical condition and able to 'play with complete abandon'. He should concentrate completely on the game and be consumed by the will to win. In performing effectively the athlete must be able to run rapidly for short distances, stop suddenly, turn or 'cut' to either side and handle the round ball with agility and smoothness. He must have an excellent sense of timing and balance and be able to shoot for a basket from awkward positions. Since this is a sport which requires fast action in close quarters, it is inevitable that some injuries will occur. The parts of the body most vulnerable to injury are those portions subjected to the greatest strain.

Dr Kimbrough in another section mentioned the basic musculo-tendinous-bone unit in baseball injuries and it will not be necessary to repeat this here. The points which are most vulnerable to injury in basketball are the feet, ankles and knees in the weight bearing extremities, and the fingers, hands and wrists in the upper extremities. Other areas of the body are injured, but much less frequently. At times these other injuries may be serious.

In our experience with the basketball team at the University of South Carolina, the most frequently observed injuries have been sprains to the ankles. There may be partial or complete tearing of the fibulotalar ligament. This is produced by the sudden inversion in plantar flexion of the foot with external rotation of the tibia on the talus. This injury occurs frequently because of rosin on shoes to assist in quick stops and turns. Sudden motions

magnify the forces applied at the ankle joint. In severe injuries the fibulo-calcaneal ligament is torn with disruption of the stability in the ankle mortice and subsequent impairment of function. Injuries to the ankles occur more frequently under the boards during the process of rebounding the ball.

The next most frequent injury has been damage to the outstretched fingers of the hand and is commonly termed a 'jammed' finger. This is very similar to the 'baseball finger' or 'mallet finger', which is produced by a complete disruption of the musclo-tendinous-bone unit. Usually in the basketball injury there is no complete separation of the tendon. Any of the interphalangeal joints or the metacarpo-phalangeal joints may be involved. The injury is the result of a compression force caused by the basketball striking the outstretched finger or thumb. Traumatic synovitis develops with swelling, stiffness and pain. Occasionally dislocations or subluxations may occur and at times a typical mallet or baseball finger may be seen in basketball. With taping or splinting, the athlete is usually able to continue playing despite these injuries.

Another injury which occurs with almost the same frequency as the jammed finger is the abrasion or 'strawberry' which develops on knees or elbows as the result of falling or skidding on the hardwood floor. Abrasions may devlop on any part of the body. Usually they are superficial and present no real problem in treatment. At times they may prevent a player from performing to his full capacity.

Injuries involving the knee are next in frequency. Here the medial semi-lunar cartilage is most often damaged. With the torsion and lateral stress applied to the knee in basketball, it is amazing that there are not more injuries involving this structure. In violent, twisting movements, either collateral ligament may be torn. When the force is continued, the semilunar cartilage or the cruciate ligaments may be involved. At times the medial collateral ligament, medial semilunar cartilage and anterior cruciate ligament are damaged (O'Donoghue's triad). These severe injuries must be treated by prompt surgical repair.

A 'Charlie horse' or contusion of the muscle often occurs and usually involves the quadruceps muscle. Muscle fibres are torn by a sharp blow from a blunt object and a haematoma ensues. When periosteum is involved, myositis ossificans may develop. The frequency and severity of the 'Charlie horse' can be diminished by adequate padding.

Fractures most commonly seen in basketball are those of the small bones of the hands or feet. Occasionally they are accompanied by dislocations. Rarely are there fractures of the long bones or of the axial skeleton.

At times a player is injured by cerebral concussion or more serious brain damage.

Most injuries in basketball can be diminished by requiring participants to report in excellent physical condition. Conditioning can be achieved by an exercise programme that begins several weeks before the basketball season. The players should have an excellent diet, including vitamins, minerals and proteins. Conditioning of the players should include running. They should run slowly at first and gain speed as strength develops. When muscular development and endurance have improved, then they should start turning rapidly or 'cutting'. The players should go through a warming up period before going 'full tilt'. It has been well established that rapid, sudden contractions of 'cold' muscles may cause serious injury to the musculo-tendinous-bone unit. A gradual warm up period before participation in a game is recommended at all times.

The areas most subject to injury should be protected against stress. The ankles are protected by adhesive strapping. The type of strapping we have used is similar to the 'Gibney boot'. With this particular type of strapping the emphasis is placed on giving support to the collateral ligaments, the fibulo-calcaneal and the fibulo-talar ligaments. These are the most frequently injured ligaments in basketball.

Before taping, the skin is prepared with Tincture of Benzoin. The foot is held in a neutral position with the ankle in slight eversion. This causes the adhesive tape to be tighter on the lateral aspect of the ankle than on the medial side. If the skin does not tolerate adhesive tape, the skin can be wrapped with gauze and the tape applied over the gauze. This is not as efficient as the taping. A light flannel wrap may also be used. Numerous methods of ankle strapping have been devised and advocated. There are rubber, elastic and leather ankle supports available but we have found these to be less effective than adhesive strapping, in preventing injuries. If an ankle is injured despite adhesive strapping an accurate diagnosis should be made and prompt treatment instituted. Stress X-ray films should be made of both ankles for comparison, to check on the amount of tilt of the talus in the ankle mortice. This method is very informative. If the ligament is completely torn it should be treated either by cast immobilization or by surgical repair and immobilization until adequate healing has occurred.

Very little can be done to prevent injury to the digits of the hand and retain the agility, smoothness of action, and function which is necessary. For that reason we have been treating these injuries as they occur and have found no effective method of preventing them. Very little, other than experience, can

be done to prevent injuries to the hands. Fortunately, they are not serious and usually cause little loss of time from the sport or from school.

The abrasions or 'strawberries' which occur either in the elbows or the knees can effectively be prevented by the use of pads. Some abrasions will occur despite these measures and each player should have a tetanus immunization series.

When a player has a weakness of the medial collateral ligament of the knee or of any other ligament, the joint should always be protected by adhesive strapping. This affords some degree of stability to the joint and reinforces the strength of the ligament so the player can perform to his best ability. To date we have found no effective method of preventing meniscus injuries or collateral ligament injuries.

In summary the most frequent injuries in basketball have been discussed. The importance of basketball players being in excellent physical condition has been emphasized. Protective measures guarding against injury to parts most vulnerable have been mentioned. Prompt treatment of injuries with anatomical replacement of separated parts is recommended.

A player has a 'bill of rights' which should make mandatory good coaching, good equipment and good medical care for all sports.

BIBLIOGRAPHY

1. *Clinical Orthopaedics*, Vol. 23, pp. 106-107; 146-169.
2. *The Trainers Bible*, 9th revised edition, S. E. Bilik, M.D., F.A.B.P.M. R.
3. *An Encyclopedia of Causes, Effects and Treatments of Athletic Injuries*, J. V. Cerney.
4. *The Journal of the American Medical Association*, Vol. 169, No. 13, pp. 1405-1436.

7

BOXING INJURIES

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I HAVE spent thirty years in the medical control of amateur boxing and although it is practically the only sport where the contestants are deliberately striking each other, the number of injuries and fatalities are relatively rare.

Boxing injuries rank eighth in frequency among sports injuries. In 4,350 contests held under the auspices of the London Amateur Boxing Association in the season 1957-1958, 137 injuries were reported. There were 42 cuts in the region of the eye, six of the lips, three of the forehead, two of the ears, three of the mouth and one of the nose. There were three fractured metacarpals, two fractured noses, sixty knock-outs and nine cases of amnesia without loss of consciousness. In addition, there were five sprains involving the metacarpo-phalangeal joint of the thumb or inter-phalangeal joints of the fingers.

EPISTAXIS

The commonest boxing injury is simple nose bleeding. This can easily be stopped during rounds by sitting the boxer upright, pinching the nares and applying a cold sponge. If the bleeding persists after the bout, plugging with adrenaline gauze or adrenaline-soaked cotton-wool usually stops it. The plugging should be removed after twelve hours. More severe bleeding may indicate obvious damage to the septum and this will mean early removal to hospital where the bones can be manipulated with Walsham's forceps before bleeding and oedema cause obstruction. Boxers subject to nose bleeding may have an enlarged nasal vein which can be cauterized.

DEFLECTED SEPTUM AND FRACTURED NASAL BONES

Deflected septum: A cartilage injury is relatively common and may cause considerable nasal obstruction; damage to the ethmoid or vomer is rare. Manipulation should be done within ten days. Often when oedema subsides

manipulation is found to be unnecessary. Boxing can be resumed after two to three months. If manipulation is unsuccessful, a submucous resection is necessary; this means the abandonment of boxing, as does remodelling of nasal contours.

Fractured nasal bones: This is rare; there were two cases in the 4,350 contests. Elevation by Walsham's forceps is indicated; boxing should be forbidden for three months.

ABRASIONS AND LACERATIONS

Abrasions: These are common on the face and skull. Bleeding should be stopped by pressure: fibrin foam applied with pressure on a pad of gauze or cotton-wool is usually successful in obstinate cases. Bathing of the wound with warm saline is followed by the application of cetrimide (1 per cent) and light dusting with sulphathiazole powder. Antibiotics should not be applied locally as they may cause sensitivity reactions.

Lacerations: These occur in about one out of every ten contests. When the wound is dry and has been cleaned as indicated above, it should be sutured to ensure sounder and more rapid healing and less disfigurement. The so-called 'cut-eye' is usually situated over the supra-orbital ridge or in the upper eyelid. In the scalp, if the wound has gone right through the thickness of the skin, suturing is essential, co-apting the whole of the cut surfaces. Elsewhere, if there is no contusion and no dead space in the depths, it can be closed by

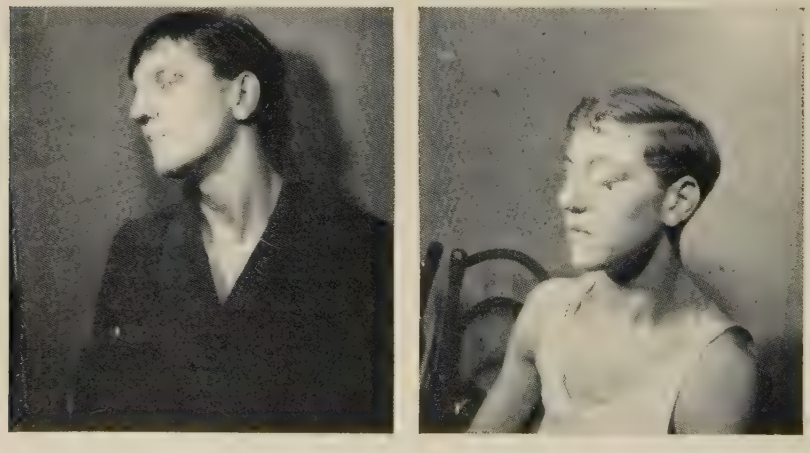

FIG. 1. Common boxing injuries: *left*; cut upper eye-lid: *right*; haematoma of upper eye-lid.

approximating the skin edges (Fig. 1). Von Horff's clips are an easy and quick way of getting the skin edges together. A gauze dressing with collodion is applied and the clips are removed after a few days. Small dry wounds can be held together and sealed with a plastic material, e.g. 'novecutane'. The most suitable suture material is silk thread. This can be obtained already threaded into small curved needles in sterile containers, and a Gillies needle-holder is most useful. Sutures should be removed within five days. The sutures can also be applied through a fine hypodermic needle. Dumb-bell plaster strips applied at right-angles to the wound are also useful. Boxing should be prohibited for at least four weeks. Most of these 'cuts' are caused by the opponent's head. Boxers should be trained to keep their heads up and avoid crouching. Referees could be stricter with offenders, and early disqualifications might lessen the number of offenders.

HAEMATOMAS

The common 'black-eye' hardly ever needs treatment, but cold applications limit the extravasation of blood (Fig. 1). A haematoma of the auricle, if seen early, should be aspirated and 1 or 2 ml. of hyalase injected. If it is seen late the clot must be evacuated, the wound plugged and allowed to granulate from the bottom.

CONCUSSION

This occurs about three times in a hundred bouts and is caused by a direct blow to the jaw, an accumulation of the effects of blows on the head, or by the head striking the floor of the ring. Most boxers regain consciousness by the count of ten and can be assisted to their corner. If consciousness has not been regained by the end of the count the boxer should be allowed to recover on the floor of the ring. His head should be supported and turned to one side, making sure that there is a good airway. On recovery he should be carried to his corner. He should not be stimulated in any way: e.g. by having his ears rubbed or smelling salts applied to his nostrils. If he remains unconscious for any length of time, he should be removed from the ring on a stretcher and sent to hospital for observation. If there is retrograde or post-traumatic amnesia (loss of memory for events before or after the knock-out), even without loss of consciousness, the patient should be treated as though he had concussion and admitted to hospital. After concusion the minimum period off boxing is four weeks. If the concussion is severe, an electroencephalogram (E.E.G.) may be advisable, but it must be done within two weeks of the

FIG. 2. Investigation of cerebral function by an electroencephelogram. Recording apparatus in position.

injury (Fig. 2). Any abnormality found would indicate a longer period off boxing. The E.E.G. is not pathognomonic: during the 1957-1958 season, seventeen boxers were referred for E.E.G. examinations after severe concussion and no abnormality was found. An abnormal E.E.G. generally returns to normal in about two weeks. The so-called 'punch drunk' syndrome is very rare. I have only seen one case in an amateur boxer in thirty years. He was an Army boxing instructor who took on all comers for eight hours a day!

We performed E.E.G.s on 100 young boxers before and immediately after boxing. 48 per cent showed abnormal slow waves without any pathological cause before boxing. It was shown that those boxers receiving most blows on the face showed the greatest number of changes. Concussed boxers

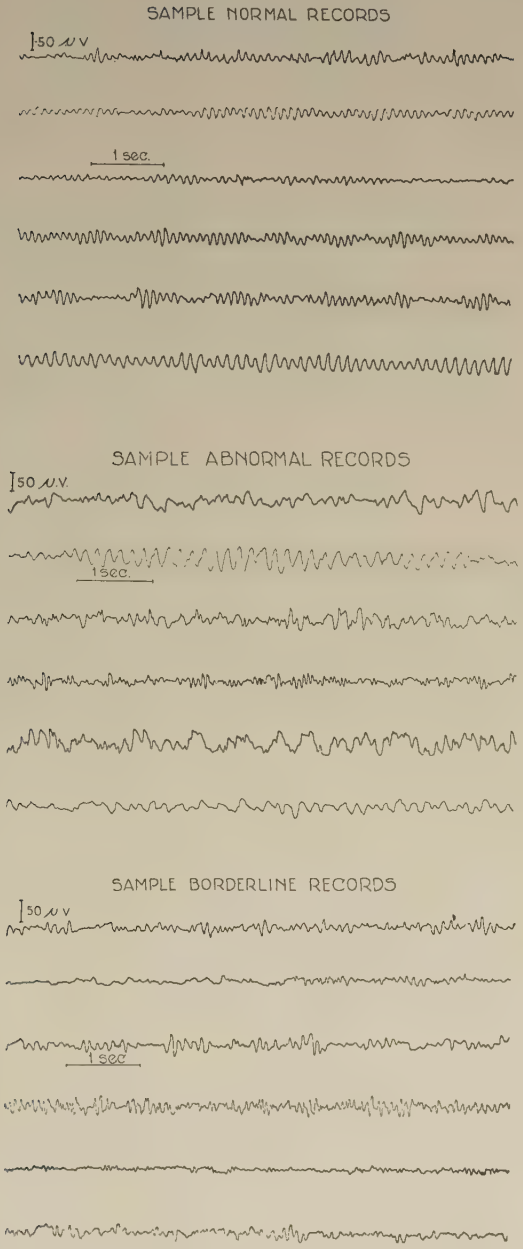

FIG. 3a, b and c. Sample of normal, abnormal and borderline cases.

showed far many less changes. The changes did not persist more than four minutes and were physiological in character.

FRACTURE OF THE SKULL AND INTRACRANIAL HAEMORRHAGE

Are rare: eight cases in thirteen years.

Fractures of the jaw are also rare. The average is about two a season. A boxer should be given first-aid treatment and, if the jaw is also dislocated, it should be reduced at once by pressure on the lower molar teeth downwards and backwards while the chin is moved upwards; he should then be sent to a maxillo-facial unit. Boxing should be forbidden for six months.

Serious eye injuries are also very rare. I have seen two cases of detached retina in amateur boxers in thirty years, but I have seen three as a result of squash rackets!

FRACTURES AND SPRAINS

The commonest fractures are those of the index metacarpal. Fracture of the shaft usually occurs within the proximal third of the bone. Any significant displacement or angulation should be corrected by manipulation under an anaesthetic followed by immobilization for three to four weeks. This is followed by active movements; painless function is restored in six to eight weeks.

The Bennett type of fracture of the base of the thumb metacarpal involves a true dislocation of the carpo-metacarpal joint. Reduction is carried out by skin, pulp, or skeletal traction for three weeks the thumb being in abduction within a below elbow plaster of Paris cast. Immobilization is maintained for a further three weeks, followed by active movements. Boxing can be resumed after three months.

Fractures of the shafts of other metacarpal bones seldom require anything more than plaster of Paris protection for a week or so. If there is angulation or shortening due to overlap, manipulative correction should be undertaken and the wrist and metacarpals immobilized for three to four weeks. Boxing can be resumed after eight weeks.

The commonest joint to be damaged is the thumb metacarpo-phalangeal joint. There may be extensive damage to the capsule with possible rupture of a collateral ligament; or a chip of bone may be avulsed. Immobilization in semiflexion for three weeks is essential, followed by active exercises. Operative repair may become necessary. Boxing may not be possible for six weeks. Sprains of the inter-phalangeal joints should be treated on similar lines.

Fractures of the limb bones and injuries to other joints are extremely rare in boxing, although I have had to replace displaced semilunar cartilages in the ring and have seen one case of dislocated hip!

Preventive measures

Many boxing injuries can be prevented by the wearing of bandages over the metacarpals, the use of gum shields, cup-protectors, jock straps and laceless gloves weighing 8 ounces (227 g.), and by having a 'sorbo' rubber covering under the canvas of the ring.

Fitness in boxing is essential. Three 3-minute rounds of boxing are more exhausting than the 4-minute mile! Roadwork is the best exercise to improve the leg muscles, which are the first to go in boxing, and to improve the stamina. Abstinence from smoking and alcohol, and early nights are indicated. In recent years boxers have become more of the slugger type. The upright stance, using a good straight left not only scores more points but is the best method of defence. It also makes less likely the 'cuts' which are more prevalent these days.

I have recently used tablets of buccal varidase to reduce the number of bruises and haematoma and to treat them, as well as abrasions, cuts and joint swellings. Two tablets are dissolved slowly in the mouth about one to two hours before the exercise, whilst one tablet is taken four times daily after meals for three days if injury occurs. The results have been most encouraging, and boxers have been able to resume boxing much sooner. There are no side-effects.

BOXING INJURIES

HENRY COOPER

Heavyweight Champion of Great Britain
Heavyweight Champion of the British Empire

HENRY COOPER, heavyweight champion of Great Britain, who began boxing at the age of twelve, has more than fifteen years' experience of the amateur and professional ring. At a recent interview he gave us his views on the injuries with which he had become familiar in the course of his career.

There was a time when knuckle injuries were frequent, whether of the thumb or of the ring and little finger. Though bandages were generally worn

under the boxing gloves, they did not prevent bruising and sometimes fractures: but the technique of bandaging has now improved. Cotton bandages, which are more rigid, have replaced crepe; and these, held together by zinc oxide strapping, help effectively to prevent injuries to the fist. Professional boxers of any eminence take no risks with their knuckles, preparing the bandage and tape as carefully for three rounds with a sparring partner as for a championship fight. Therefore this sort of injury although still common can often be prevented by minute attention to detail.

FIG. 4. A close-up shot of a pounding on the ropes.

More common, and more serious, are cuts in the skin around the eyes. Deep cuts are usually opened in the first instance by a blow from the opponent's head; but in later bouts, the damaged area may be split by a punch. Many fights are ended prematurely as the result of a clash of heads. Such incidents seem to have become more common in recent years, perhaps because the modern boxers crouch more than their predecessors, constantly moving the trunk and head, perhaps because referees are less inclined to allow a deep cut to be further damaged. (Henry Cooper's father tells him that cuts were as common in the old days of the Blackfriars ring, but the referee seldom stopped the fight).

The classical sign of the old pugilist, the cauliflower ear, was the mark not of the third-rate but of the clever boxer who 'slipped' all the punches, being caused rather by constant friction than by direct blows. Ribs are frequently bruised, sometimes fractured, by body punching; and occasionally a rib may be separated at the costo-sternal junction.

These are all direct injuries: there are also some which, though they are caused by direct violence, are indirect in the sense that they do not result from contact with the opponent: 'burns' of the skin, for example, when a boxer slides along the ropes, or damage to the back of the head against the floor of the ring. In America the boxing authorities are said to be experimenting with thick foam rubber or plastic padding of the boards.

There are ways in which a boxer can prepare himself to prevent or to minimize injury. Henry Cooper takes particular care to see that he is at his best weight. If he is 2 lbs. overweight, he finds that his reactions and movements are comparatively slow. He keeps fit at all times, being abstemious in every sense, and goes into strict training six weeks before a fight. On the day, his main meal is a protein one—a large steak with salad, several hours before the bout. He attaches great importance to 'warming-up' before violent exercise, so that his muscles are ready for the demands to be made upon them; and, as the best insurance against injury, he tries to cultivate alertness, physical and mental, so that he can anticipate and react both in defence and attack.

8

INJURIES IN CRICKET

N. VERE-HODGE
F.R.C.S.
Cambridge University Crusaders and Essex County

FORTUNATELY, in cricket injuries are relatively infrequent. The more unfit and older the player the more likely the chance and severity of these injuries.

There are three main types of injury:—

(a) The usual strains and muscle damage produced by sudden fast movements.

(b) Contusions and fractures caused by being struck by the ball.

(c) Special injuries peculiar to cricket.

SUDDEN FAST MOVEMENTS

The sudden fast movements which are particularly likely to cause injuries in cricket are the twisting of either knee when batting or wicket-keeping, which may result in the strain of one of the ligaments of the knee joint or tearing of a 'cartilage'.

Slipping on a wet wicket when running, may cause a partial or complete rupture of the ham-strings or tendo-Achilles.

Sudden twisting combined with bending when fielding may cause a lumbar strain or even a 'slipped disc'.

DAMAGE FROM THE CRICKET BALL

A cricket ball weighs $5\frac{1}{2}$ ounces and can travel at speeds estimated to be 80-90 miles an hour, therefore serious damage can result from being struck by the ball. One of the commonest injuries is to be struck on the head when batting either directly or by a ball deflected off the bat. The precise injury naturally depends upon the site of the blow but it may result in concussion with varying degrees of unconsciousness and, in extreme circumstances intracranial damage and death. Wearing a cricket cap which has padding over the forehead may soften the blow or deflect the ball thereby diminishing the damage to some extent.

It is most important that those who require to wear glasses should use the unbreakable type of lenses to avoid fragments of glass damaging the eyes.

Fracture of the nose and teeth occur occasionally when wicket keeping.

Blows over the chest may result in marked shock and fractured ribs and blows over the heart may be dangerous. Fortunately the shock is usually of limited duration, but may well upset the concentration of a batsman.

Blows over the scrotum can cause much pain, shock and bruising. There may be damage to the testicles of varying extent. For the prevention of this type of injury it is always essential to wear the special aluminium protector for batting or wicket keeping.

Blows on the fingers when batting are probably the commonest injury and frequently cause fractures in spite of using batting gloves. When fielding or catching, any misjudgement of the flight of the ball may well result in the fracture of a finger or fingers.

The legs, being largely protected by pads when batting, are seldom seriously injured but the feet are often hit by a full pitched ball, which may result in severe bruising or even a fracture of part of the foot or toes.

FIG. 1. There is no doubt about the strains imposed on a bowler.

When fielding, any misjudgement of the ball on the ground may result in bruising of the front of the shins.

SPECIAL INJURIES PECULIAR TO CRICKET

Certain special injuries peculiar to cricket are not uncommon.

1. *When bowling.* A strain of the thigh muscles in the groin can occur especially at the beginning of the season or when bowling on very hard ground.

An acute or chronic condition in the bowling shoulder, called supraspinatus tendonitis, which is produced by repeated pinching of the tissues between the acromion (the roof of the shoulder joint) and the greater tuberosity of the humerus (the upper part of the arm bone), as the arm is brought over the top in delivering the ball. This pain occurs during or after bowling and may be so obstinate that only operative treatment (to remove the roof of the shoulder joint) will allow bowling in comfort. A mild degree of this condition may be relieved by rest and injections into the damaged tissues with one of the steroid preparations (an offspring of cortisone).

Simple bruising or even a fatigue fracture of a metatarsal bone of the left foot caused by the great force with which it is thrust down on to the ground in the process of delivering the ball (assuming the bowler is right handed, the reverse applies to a left handed bowler).

A chronic splitting of the skin on the side of the main finger which spins the ball. Protective strapping cannot be applied to the fingers when bowling as it would facilitate a greater degree of spin being applied to the ball which would be an unfair advantage. The best way to protect the skin of the fingers from cracking is to keep it soft by rubbing in lanoline. Spin bowling for many years can cause repeated strains leading to osteo-arthritis of the end joint of the spinning finger or fingers.

2. *When fielding.* Throwing the ball can cause a recurrent strain of the brachialis muscle above the elbow. This can only be permanently relieved by learning to throw with a different action.

3. *When wicket keeping.* Repetitive damage to the fingers and thumb is frequent and in the course of time may well lead to instability of the joints of the fingers and thumb with osteoarthritic changes in them.

The ball can force the fingers apart and split the skin between the fingers extending into the palm. All wicket keeping gloves should have good padding over the palm, with strong rubber finger stalls inside the fingers and partial webbing between all the fingers and thumb.

4. *When batting.* The chief hazard is being struck by the ball. The possible effects of this have already been described.

INJURIES AT CRICKET IN A PLAYER'S VIEW

DENIS COMPTON
C.B.E.
Middlesex and England

THE English game of cricket is fully comprehensible only to those who have played it from early youth: but it is not necessary to understand all the subtleties in order to see that a game in which a bowler hurls a hard leather ball at a batsman is likely to result in occasional injury. Some cricket injuries—during a test series for example—become affairs of national importance which are followed by an anxious public every day. The country's hopes and fears were at one time focussed on Denis Compton's knee. In a long and illustrious career he has seen and taken many hard knocks; and he has given us the benefit of his experience in an interview which we paraphrase below.

To a question on direct injuries he replied that batsmen are the players most liable to be struck by the ball. Bruises, whether of the back of the hand (despite padded gloves), the ribs or the elbow are common; and the bone is often found to be cracked. The leg pads give essential protection, but, because they are a compromise between the opposite needs of defence and attack, are not really satisfactory for either. The top of the thigh above the pad or the calf behind the pad may be badly bruised. Sometimes, when a fast bowler is in full cry on a lively pitch, the ball may strike the batsman's face. (Here he recalled the time when a ball from Lindwall opened a cut on his face which needed a row of eight stitches). One of the most painful and incapacitating bruises is that due to the impact of the ball 'full pitch' on to the top of the foot.

Wicket-keepers have to face almost the same risks as the batsman, and hand injuries are so common among them that one time it was possible to recognise a wicket-keeper by looking at his hands. Now that all badly bruised areas are X-rayed, breaks in the finger bones are spotted and properly treated, so the distortions no longer occur. Fielders get their fingers bruised and sometimes broken when they stop a ball travelling fast in the air, or

damaged fingers and toes when the ball hops over bumpy ground. The very close leg-side fieldsmen seem to be in great danger of being hit by both bat and ball, but in fact are not more frequently injured than the others: slip fielders to fast bowling often get painful blows on the finger tips. Anyone who has experienced a 'topped' slip catch knows how painful it can be.

There are all sorts of indirect injuries and all players are sometimes affected. Batsmen and fielders often strain the groin muscles when they make a sudden, violent turn; batsmen develop thickening of the right forefinger (if they are right-handed) due to constant friction with the handle when they are in good form and spending long periods at the wicket; fieldsmen injure tendons when they throw hard, especially if they are off balance. But the bowler is the one most often affected. Fast bowlers have to take great care to 'work-up' gradually when the season starts; and as the weather is so often cold at this time, they must combine the general 'work-up' with a progressive 'warm-up' on the particular day or they are liable to get muscle strains and tears, particularly of the abdominal region. Medium-pace bowlers put great strain on the low-back and buttock muscles, and if these are not trained and ready for the effort, they give trouble on the side opposite to the bowling arms. Twists of the knee and ankle may be fundamentally the result of an unsound bowling action, but the immediate cause is often unevenness of the ground on which other bowlers have been banging down their feet. Slow bowlers have their own troubles, particularly soreness and thickening of the spinning finger or inflammation of the tendons of the shoulder muscles.

When asked about his own famous knee injury he said it was originally damaged on the football field not at cricket: but he has had ample opportunity to form opinions upon how cricket injuries can be avoided. First in importance he places 'working up ' gradually at the beginning of the season, and 'warming-up' on the day. These are true preventive measures, aimed mainly at the indirect injuries. A bowler who, despite careful preparations, consistently injures himself ought to consider changing his style, even if the change has to be a really fundamental one. The direct injuries are less easy to prevent. But, in general, accidents happen to those whose concentration has wavered, whether by inattention or fatigue: and the best insurance against injury is a conscious and mental alertness until stumps are drawn.

9

THE FENCER AT RISK

R. PARFITT
M.R.C.S., L.R.C.P., L.D.S., D.M.R.T., F.F.R.

British Epee Champion, 1948, 1950
Olympic Epee Finalist, 1948
Chairman, Technical Sub-Committee,
Vice President, Amateur Fencing Association
Corresponding Member, Technical Commission,
Fédération Internationale d'Escrime

Sudden, Achilles his broad sword display'd
And buried in his neck the reeking blade.
Prone fell the youth ; and panting on the land
The gushing purple dyed the thirsty sand.

HOMER: 580 B.C.

FENCING is not a dangerous sport, but if one bears in mind its descent from the bloodthirsty brutalities of sword fighting, and the somewhat more elegant but equally bloodthirsty art of duelling, it is not surprising that a few accidents happen. Some have proved fatal.

The injuries group themselves into three kinds. The first includes those inevitably accompanying any activity where the whole body or part of it is moved frequently and with maximum acceleration over short distances. Like any other movements the basic laws of mechanics can be applied to them.

$$f = m\,a$$

where:—f = force
m = mass
a = acceleration.

It makes for clearer thinking when it is realised that force results in acceleration, i.e. a change of speed, and that constant speed needs little force, only enough in fact to overcome losses such as friction in the situations now to be discussed.

Fencing Movements

A fencer's normal stance is upright, with knees well bent and feet at right angles separated by about twenty inches. The feet are upon a line running the length of the Piste.[1] He advances or retires along this line. It follows that his

base is long, and therefore stable, along this line, but short and therefore unstable across it. To avoid staggering or falling sideways the feet must remain flat on the floor as much as possible.

An advance is made by lifting the front foot just clear of the floor, moving it forward and replacing it heel first. The rear foot follows. The body moves

FIG. 1. To maintain balance the fencer's feet should be as flat on the floor as possible.

forward at the same time so that its centre of gravity remains above a point about midway between the feet. To retire, the procedure is reversed.

A lunge consists of the same movement with the front foot but the rear foot stays where it is. The rear knee is extended sharply and the body bent somewhat forward. The arm is straightened at the beginning of the movement with the result that the point is moved forward with maximum acceleration. The hit should land while the point is still accelerating.

There is only one other way of approaching an opponent. This is known as the flèche. It is the fastest attack but pays for its speed by leaving the attacker poorly balanced and probably too close. He merely leans his body forward and straightens both legs, the rear one first, as he straightens his arm. At the moment when the point should land the body and forward leg are in line, almost straight, and at about 45° to the floor. The rear foot has left the floor and is crossing the front foot. The weight is on the front foot which at this moment is transmitting maximum accelerating force. If the rear foot is not brought up to cross some distance in front of the other the fencer will fall flat on his face, and whether he scores or not will look a clumsy fool. The forward momentum (Mass X velocity) may well be so great that the braking action of the rear leg, now having reached the floor well in front, may not be enough to stop the forward action and the legs are again crossed so that another pace is taken. The flèche may continue as a run into the opponent, or more correctly leaving him on the left.

The arm besides forming the thrust already described as part of the lunge or flèche makes many other movements. The only one we need consider is the parry. This involves moving the arm and blade in the shortest time possible into one of several positions, each of which closes the line to some part of the defenders target. It is, of course, hoped to meet the attacking blade in the process, and thereby divert the attack. In the 'parry seconde', for example, the blade is moved from a position of about 30° above horizontal to just below it, the hand and forearm being simultaneously pronated. The movement is equally abruptly stopped in this position, or should be. It is not a wild swipe intended to strike the attacking blade downwards. The blade should stop in the correct position whether the opponent's blade has been found or not.

Mechanics of small movements

A plot of speed of blade movement against time may be represented as the curve shown in Fig. 2. At A the parry starts, with maximum contraction of the pronators of the forearm and some extension at the elbow. The movement is, of course, supported by co-ordination of almost every other muscle in the

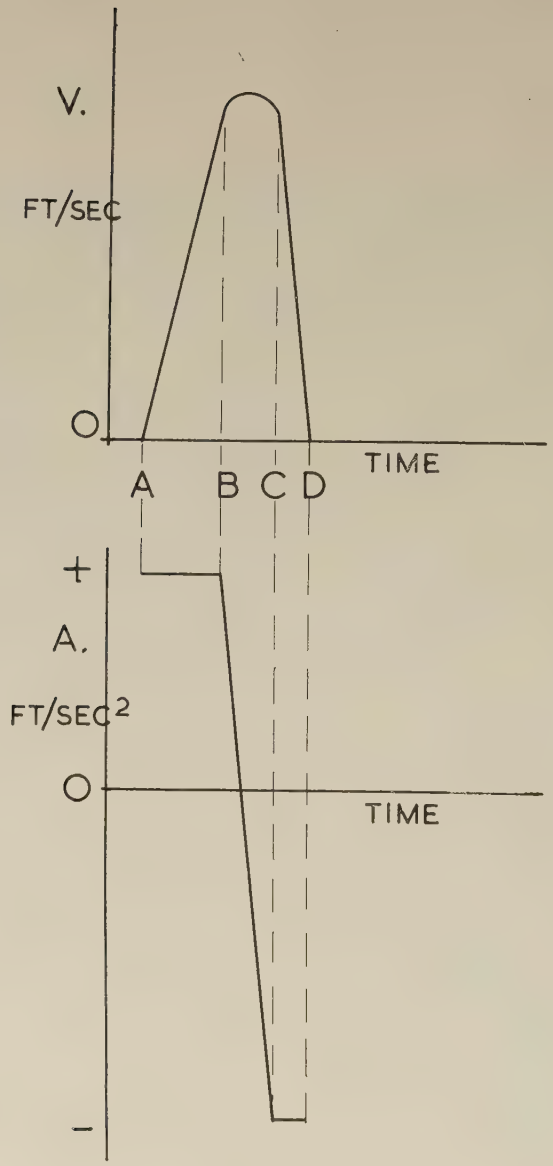

FIG. 2. Blade velocity, upper curve, and acceleration, lower curve during parry Seconde.

body to some extent. The blade accelerates at its maximum until a point is reached at time B. Now, whether the attacker's blade has been met or not, if more than half the distance has been covered, an opposite force is applied to the weapon to prevent overshoot converting the parry into a swipe. Those muscles previously relaxing to allow the movement so far, now must strongly contract against movement and the force they exert is probably greater than that of their antagonist's which initiated it. Between time B and C the force is being reversed and therefore there must be a moment when no force is exerted at all. The velocity at this moment is constant and at a maximum. Maximum effort is then made in the opposite direction against the movement of the arm and blade; and for this reason is probably greater than the original muscular effort of the first part of the movement. This muscular effort at any moment is equal to the slope of the velocity graph of Fig. 2 upper curve, and is shown in the lower curve. Its direction of course reverses. An example will help to make the point clear. If one runs towards a stationary swing in order to give the passenger the strange pleasure of behaving like a pendulum, both pusher and pushed will feel a jar: but if one meets a moving swing at its lowest point, the jar is considerably greater.

Injuries due to reversal of force

It is these sudden reversals of direction and force, which cause injuries such as the 'pulled muscle', 'strained back', etc. to which fencers are liable. Tennis elbow is a common example. To those who have fenced with a tennis elbow it is noticeable how the checking of certain movements is painful, the worst being the completion, but not the start of parry seconde.

Injuries to the knees are few, due presumably to there being little tendency to twist. Hip joints are well able to look after themselves although one fences in a position which invites dislocation, should sudden excessive downward force be applied. The best fencers do not leap heavily upon one another's shoulders and dislocation is exceedingly rare. Intervertebral joints, or the muscles supporting them, give trouble from time to time, the injury being called a 'strain' or a 'disc' according to the preference of its owner.

The shoulder, being an unstable joint, occasionally suffers similar injury, mainly among sabreurs, whose shoulder movements are wider than those whose weapon is the foil[2] or epee.[3] The injury results in the supraspinatus syndrome, which will keep a patient out of first class fencing for months on end. Primary dislocation of the shoulder is seldom the result of fencing although a fencer has been seen wearing a harness in the hope of controlling a recurrently dislocating shoulder. It was not very effective.

Injuries due to impact

The second class of injury is due to impact. It has already been mentioned that the heel should be kept upon the floor and is the first part of the foot to meet it during a movement. The ball of the foot, by fine co-ordination, takes most of the jar, but a bruised heel is a common minor injury and is disabling as far as effective footwork is concerned for the few weeks it takes to resolve.

Any fencer may slip and fall or be driven back over the end of the rear line to crash into the wall of the Salle.[4] The injuries resulting are not specific to fencing. Similarly a fencer may be knocked down by an opponent who flèches him dangerously. This, by the rules, is barred following a fatal accident to be described shortly, and results in a warning from the President.[5] The writer has seen a blow accidentally delivered to the ribs by the coquille[6] of an epee. The coquille defied all efforts to straighten it, but no ribs were broken. On occasion a point at epee lands heavily on the mask which is driven back into the wearer's face, smashing his glasses if he wears any, and leaving an imprint of the mesh upon his skin. To avoid this the mask should fit snugly and rigidly on the forehead, and the chin should fit into a suitable depression. The whole should be adequately padded.

At Sabre[7] the main object is to strike ones opponent. A correctly made cut is delivered with the distal few inches of the blade which carries on in a slicing action. The blade is sufficiently light for the blow to be negligible. The sound it makes resembles a not very loud snap of the fingers. If it sounds like a carpet being beaten it is ill judged as far as distance is concerned and is too heavy. It is also painful and infuriating to the recipient. He is put in mind of being at the receiving end of a cane, and indeed may have a bleeding wheal to show for it. It is particularly painful if a cut of this sort lands on the shoulder as the blade bends and seems to retain most of its energy, like the lash of a whip, until the extreme end meets the jacket. Such movements are rarely if ever seen in better class fencing. They are easy to parry and it is to no one's advantage to make them. The Achilles heel of the Sabreur is his little finger, which will be hit, sometimes heavily, by a cut to the wrist if the parry is incorrectly made. On occasion a fractured phalange results.

Injuries due to penetration

The most serious injuries and all the fatal ones are caused by penetration and the mechanics of this disaster should be clearly understood. The first question to be answered is:—Is there enough force behind an attacking move to cause penetration? From the preceding description of the lunge or flèche there obviously is. Almost the entire kinetic energy ($\frac{1}{2}$ mv^2) of the

fencer is available at his hand which grasps his weapon firmly, rather as a golf club or even a bradawl is gripped.

Why then does the point not always penetrate? There are of course good reasons. One is that only a fraction of the total force is transmitted by the blade. Firstly because it bends and secondly because it rarely hits exactly true and being thus at an angle the force it can apply is immediately reduced. Another, and in fact the main safety factor is the thick canvas protective clothing worn, or the steel mesh in the case of the mask.

For anything solid to penetrate a material two factors are concerned. Either its shear stress or its tensile strength must be exceeded. In simple terms it must either be cut or broken, perhaps both. These two are measured in force per unit area. For the vast majority of substances shear stress is less than tensile strength and the not surprising fact emerges that it is easier to cut a material than to break it. It does not follow though by any means (as will be seen later) that tensile strength can be ignored. If one tears ones clothes on barbed wire the damage is obviously a tear and not a stab.

If these stresses whether tending to cut or break a material are measured in, say, lbs. per sq. inch, then the area of the end of the point of a weapon is all important, and is constant for any particular direction unless the blade breaks. Breakage is frequent and on occasions the broken end is sharply pointed. The remainder of the weapon is thus shorter, stiffer and pointed. Accidents from this cause are, however, rare because the breaking of a blade tends to deflect the rest of the weapon. The breakage also immediately becomes apparent to the fencers because of the jar and the sound it makes; and the attacker immediately and reflexly stops. He endangers his opponent only if he carries on for a distance greater than the length of the blade broken off. If only a small piece has broken off, then the blade remaining is almost as flexible as it was previously, although, of course, the end may be sharp.

The dangers in connection with protective clothing are well recognised. Canvas when worn or threadbare or rotted by sweat and much laundering, becomes friable, and so does the stitching at the seams. Masks tend to rust, especially where water condenses from the breath over the lower half. This half tends to be at an angle to the attacking blade which thus tends to glance off, and the danger is not very great.

The mechanical factors having been roughly outlined, it remains to be seen how one or more of the safety factors have been found wanting in accidents which have happened.

A fatal accident

The writer took part in the World University Championships in Paris in

1937. In his pool,[8] René Monal of France, who was left-handed, was fencing Haro Oliva of Mexico. Monal frequently attacked with a flèche, without checking his movement or running well clear of his opponent. One defence against the flèche is the stop hit. Here the defender straightens his arm, closing if possible the line of attack, and hopes thereby to place his point upon his opponent before he himself is hit. Although on paper it sounds a move unlikely to succeed, it should be remembered that the flèche is made often from out of distance and although a fast attack has further to go than the lunge. The stop hit is a planned move made as the flèche starts. It is not a wild stab made with shut eyes and a prayer on the lips.

Monal made his flèche as described and Oliva's stop landed just below the middle third of Monal's left clavicle. The blade broke near the point, Monal continued his flèche and was fatally wounded.

The factors concerned here were:—

1. A left hander against a right hander. This places their respective targets face to face so to speak. When a right hander fences another right hander the blades tend to be more oblique.
2. The dangerous flèche.
3. The fragile blade.
4. The thin jacket.

The tragedy was investigated by the Fédération Internationale d'Escrime. The rules concerning dangerous flèches were revised and ever since have been strictly enforced. Nothing further was done as far as is known.

Another fatal accident

Some fourteen years later another accident occurred during the World Championships in Sweden in 1951 and I am indebted to C. L. de. Beaumont, Esq., O.B.E., President of the A.F.A. for permission to quote his description of it in Modern British Fencing, Vol. II:

> 'A bout between Mogens Luchow, the Danish winner of the 1950 World Championships, a tall left hander and Capt. Vartia was in progress. Vartia had launched several successful flèche attacks and Luchow was waiting to stop him. On the next attack Luchow's stop hit landed on the under side of Vartia's upper arm about six inches from the armpit; the point penetrated the jacket and slipped along to the armpit. Luchow told me later that he felt no particular opposition to his blade which did not break and he thought he had merely penetrated the jacket until he saw blood on the blade.'

The blade had, in fact, passed through the right lung and mediastinum into the left lung. Vartia died within the hour. The F.I.E. enquiry established that the jacket was far too light for epee and that it was not reinforced in the axilla. The plastron was then introduced and has since been obligatory.

The plastron is a piece of protective clothing resembling a waist-coat but having in addition a short sleeve covering the upper part of the sword arm and the vulnerable axilla. It is worn under the jacket and by the regulations must be made of at least two thicknesses of hemp and must not be integral with the jacket. By this latter requirement it is hoped that a point, sliding along the upper arm after having penetrated the sleeve, will catch on the plastron and push it before it into a rumpled wad of cloth, not anchored, and therefore not exposed to any tensile stress.

Analysis of the accidents

Here then were two accidents happening under almost identical common fencing manoeuvres. There are two reasons why the point is so potentially dangerous when a left hander meets a right hander and when a point lands in the axilla. First the axilla is facing the attacker more squarely, as already

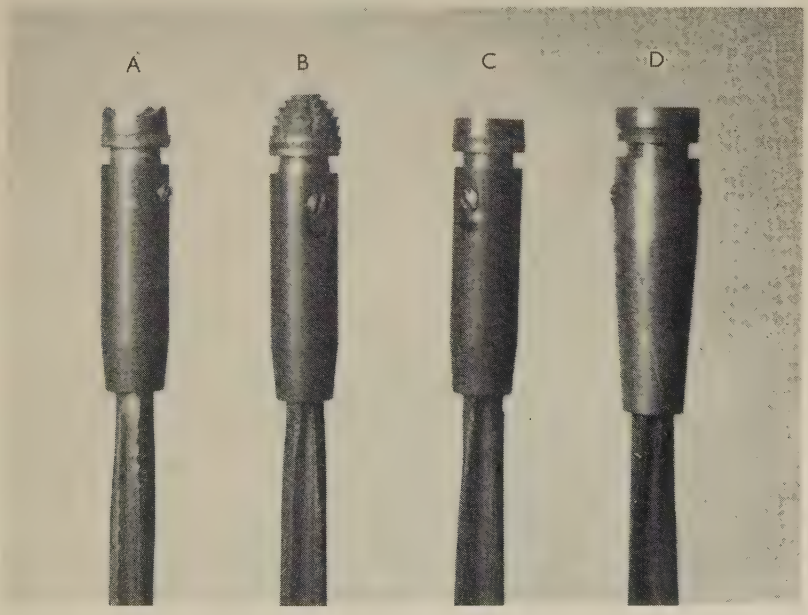

FIG. 3. Different types of epee points.

mentioned, so that the point has less chance of glancing off and, much more important, the blade is prevented from bending in one direction by the arm of the fencer hit.

Luchow's blade was in fact prevented from bending both upwards by Vartia's upper arm, and downwards by the fact that it was inside his sleeve. The epee blade in cross section is a flattened hollow ground V, and resists strongly any sideways bending stress.

The epee point in use at the time of both the accidents was 7 mm. in diameter and had four sharp peripheral blades (Fig. 3A). It was never a popular point as it always cut a thread or two of clothing if landing at all obliquely, and thereby increased ones maintenance costs.

After the 1951 accident the pineapple point, shown at B in Fig. 3, was introduced. With much surprise it was observed that this point would occasionally go through a mask. It was still 7 mm. in diameter. The mechanism of this type of accident is somewhat different. Very often during a thrust the hand is rotating somewhat and this may continue after the point has landed, for perhaps 20° or so. Likewise, as the blade bends, again the point rotates in another plane. The pineapple point will be seen to carry many teeth upon it. These are of such shape and size, as, exceptionally, to catch between two adjacent wires of the mask. The rotating action levers the two apart, and, with the unfortunate combination of a poorish mask, direction, rotation and continuation of forward pressure the point may go through.

The mesh of the mask was therefore made a little stouter and the point abandoned in favour of the flat ended point, still of the same diameter, shown at C in Fig. 3.

Once again to the astonishment of many, the writer included, it was found that clothes were still being torn and this point was evidently still dangerous.

All points are of hardened steel and although this one is ground flat at the end it is not radiused at the edges. There is thus a right angled knife edge at the periphery. If, with the object of rounding off this sharp edge, the point is pressed upon a piece of emery paper so that the blade bends, and the whole weapon is rotated as though using a brace and bit, the first result is that one drills a neat hole through the paper.

The most recent regulations (1961) demand a larger point diameter of 8 mm. (Fig. 3D). This in my opinion is not a particularly happy step. The length of the cutting edge of the 7 mm. point is $7 \times \pi$ and of the 8 mm. point $8 \times \pi$, i.e. 3·14 mm. more but with very little change in its curve. The 8 mm. point is however a little heavier. This tends to make the blade top heavy and its dynamic balance is a little upset. It can be corrected by making the blade a little stiffer, and in fact the regulations have also demanded this.

So, one is left with, once again, a nicely balanced weapon but stiffer and heavier and therefore capable routinely of delivering more force for a normal hit.

More analysis

Closer analysis is evidently needed of the stresses around the area where a point lands, and once again we must consider how a solid object can force its way through a piece of canvas.

Consider first an epee driven with its blade perpendicular to the canvas of the jacket. It bends like a bow but the force exerted remains perpendicular to the canvas along the imaginary string of the bow. The graph in Fig. 4

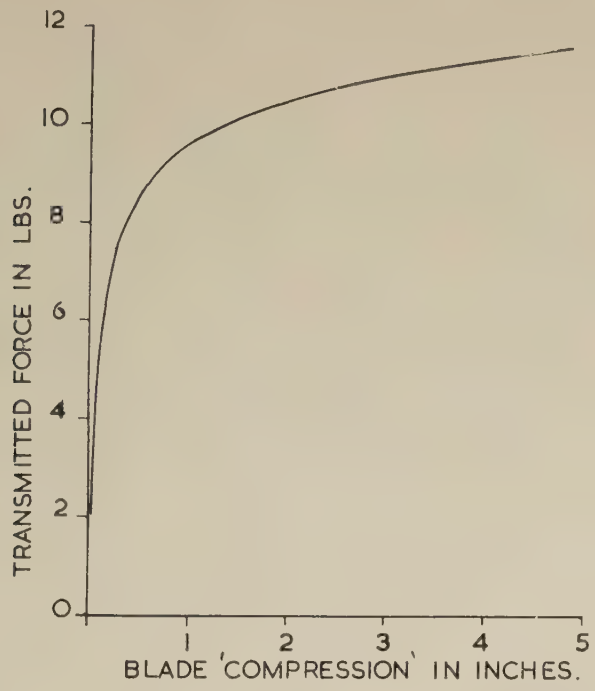

FIG. 4. Force against the fencer hit, as the blade bends.

shows that the force against the cloth increases rapidly as the hilt is pushed on towards the canvas causing the blade to bend, until about $1\frac{1}{2}$ inches 'compression' of the blade has taken place. Here the force on the canvas is about 10 lbs. A further three inches, however, increases the force only to $11\frac{1}{2}$ lbs.

For a hit landing squarely, then, the first 1½ inches or so of the forward movement are the most important. No injuries are ever caused by any of the four points shown in the photograph when they land squarely on the target. In the case of the Monal accident, the point landed squarely and broke away. The continuing flèche caused a sharp ended broken blade to land equally squarely. The shearing stress (inversely proportional to the area of the now sharp point) was more than enough to pierce canvas and human tissue.

The graph in Fig. 5 relates the angle at which the point is to the target for

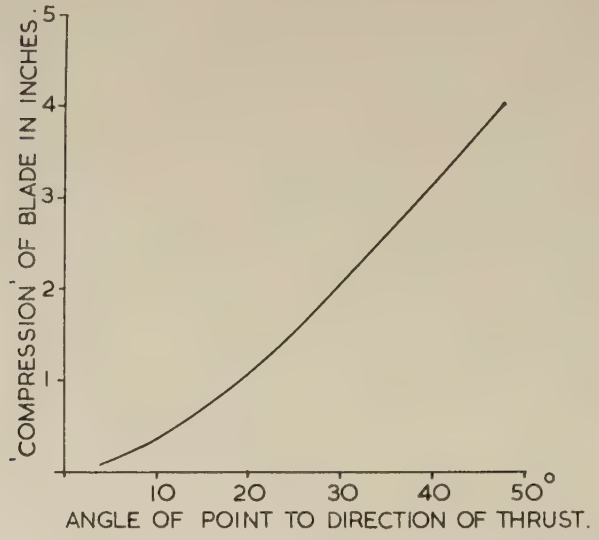

FIG. 5. The angle of the point as the blade bends.

a squarely made hit as the forward move continues. It is first, of course, a right angle and decreases almost linearly with the distance of the 'compression' of the blade. Dangerous hits thus land obliquely. The hit on the under side of Vartia's arm must have arrived at an acute angle with his arm.

Forces about the point

We must now analyse the forces about the point when it lands at an angle to the canvas. Suppose, Fig. 6, the hit with force F at any given moment, is at an angle Θ. This force obviously tends to push the area hit (D) towards A and also to move D towards C. In simpler terms the canvas if anchored at B tends to be stretched towards A and dented towards C. The magnitude of these two

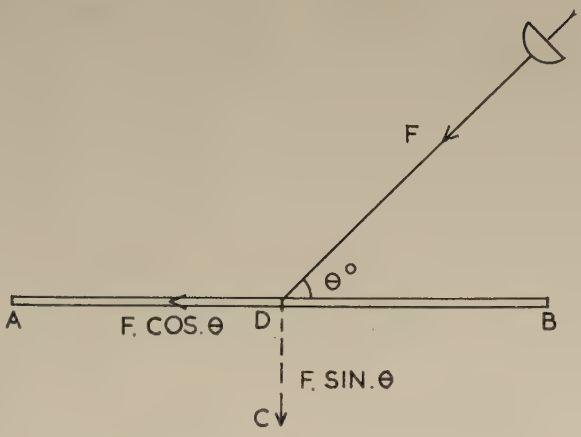

FIG. 6. Analysis of the forces about the point.

forces is easily found if the angle of θ of the attack is known. The force towards A tending to tear the canvas is F Cos θ and that tending to penetrate it is F Sin θ. Whether these will cause the point to go through depends upon their magnitude and ratio, the shape of the point and the way the blade bends; and of course, the strength of the canvas. Although, whichever way the blade bends the force F is about the same for any given degree of bend, the point will be tilted one way or the other, and therefore present a different aspect of itself in the two cases. Which is the more dangerous? Obviously the direction tending to make the bend of the blade lie flat on the canvas, because, when it has reached this position it cannot bend further and the original force of the thrust no longer follows the curve of Fig. 4 but increases very greatly.

It is common knowledge that once a tear is started in cloth it is easily continued. F Sin θ is the force tending to dig into the material and is thus all important for, if it is applied by a short length of sharp edge, it may perhaps cut a thread or two and the stretching Force F Cos θ will do the rest. What are the circumstances making F Sin θ most dangerous?

It would seem logical to examine the two situations where the transmitted force F of a thrust is about maximum at say 10 lbs. (Fig. 4). There are two positions because, as already stated, the bend of the blade may be towards or away from the canvas. The point, ending as it does in a flat surface, must be turned through 45° for its edge to be most favourably placed, i.e. cutting directly into the canvas. From the graph, Fig. 4, the compression will be $1\frac{1}{2}$ inches for a 10 lb. thrust and from the graph in Fig. 5 the angle between the

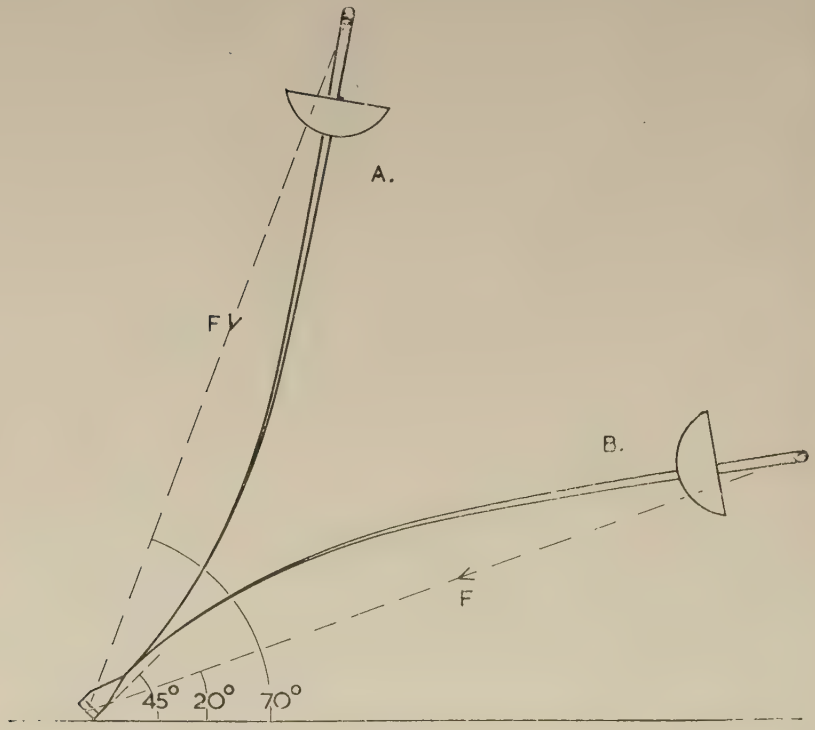

FIG. 7. The two angles for a 10 lb. hit to make the edge of the point cut directly into the canvas.

point and direction of the thrust will be 25°. We now know all the relevant angles which have been marked on Fig. 7.

For position A the tensile stress tending to tear the canvas is F Cos 70 = 3·42 lbs. and the shear tending to nick a thread or two is F Sin 70 = 9·40 lbs.

In position B they will be:

$$\begin{aligned} \text{Tensile stress} &= F \text{ Cos } 20 = 9{\cdot}40 \text{ lbs.} \\ \text{Shear} &= F \text{ Sin } 20 = 3{\cdot}42 \text{ lbs.,} \end{aligned}$$

and as one might have guessed tension and shear have changed places. All these measurements were made upon an epee used by the writer in the 1952 Olympic Games. They will vary to some extent from epee to epee.

Position A is the more dangerous, where the shear is 9·4 lbs. and the tensile stress a mere 3·42 lbs. The figures assume that the canvas stays flat, but,

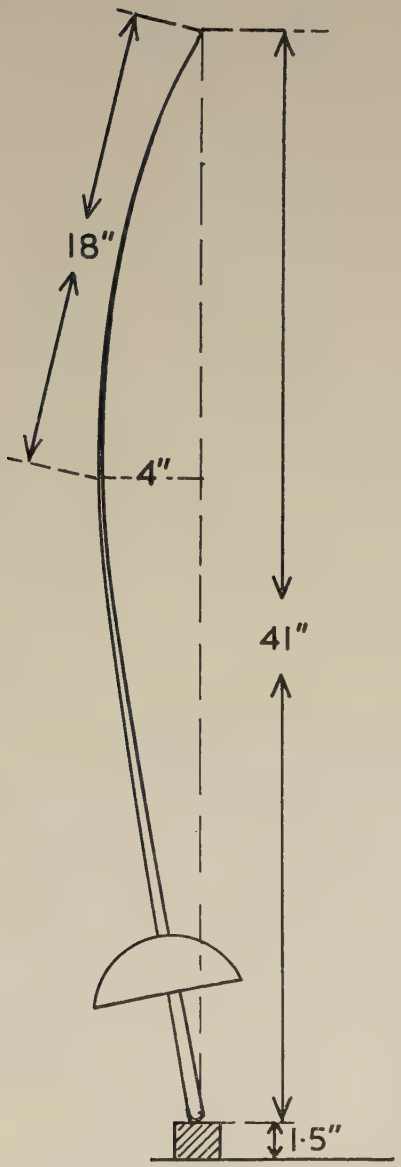

FIG. 8. The maximum bend of the blade for a 10 lb. hit.

seeing that it rests upon the fencer and that its anchorage is its own rigidity and fit, it can dimple or move in an unpredictable way. Nevertheless the mechanical principles stand. At this moment neither position is in the least dangerous. In fact fencing consists largely of an exchange of just these hits.

Danger arises in position A when further forward movement, or indeed any relative movement between the fencers, causes the bending of the blade to be impeded. Here we need another measurement from the epee from which figures have already been taken. We have already found that at F = 10 lbs. there is a compression of $1\frac{1}{2}$ inches. Further measurement shows that the maximum bend of the blade is 4 inches from the direction of the force F (i.e. the imaginary bowstring) and occurs 18 inches from the point. (Fig. 8.) The distal part of the blade is lying on some part of the fencer hit and may thus be assumed to be rigid. The rest of the blade is the thicker part and is practically rigid by manufacture. We are thus left with a simple pair of triangles of forces, (Fig. 9). PQRS with a common side QS. The forces are proportional to the lengths of the sides which we know by measurement, and are marked both in magnitude and direction. If W is the force exerted on the fencer hit by the bend of the blade, the whole system now acts as a rigid strut. Suppose W to be the not unreasonable force of 4 lbs. There is now an additional thrust upon the canvas hit and its tensile component is 18 lbs. This is in addition to the previous tensile component F Cos θ of 3·42 lbs. The shear is not increased because the blade is lying flat.

A safer point

All this burdensome middle school mechanics will be wasted unless we can deduce from it a safer point. This we can do, and it is obviously that point which presents a flat surface to the canvas irrespective of the angle of the blade. This can be achieved only in the case of a sphere of infinite radius! The safest point for practical purposes is thus one ending in a hemisphere of the largest radius possible. If this is introduced it will be an irritating point to fence with as it will continuously be glancing off the target without scoring.

All points, whatever the shape of the end, as may be seen in the photographs, take the form of a small plunger which must be depressed against a spring. This spring by the rules must withstand 750 gms. (about 1·65 lbs) before allowing the electrical contact in the point to close and so cause the apparatus to register the hit.

Conclusion

Two fatal accidents and the events leading to them have been described, and another occurred recently when a broken blade pierced the mask. The

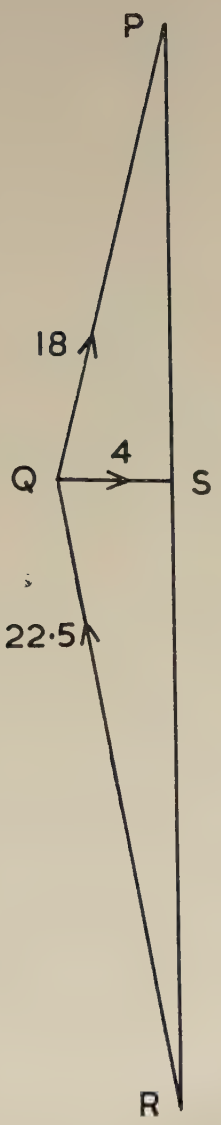

FIG. 9. Triangles of forces when the bend of the blade is impeded at Q.

F.I.E. is at the time of writing preparing a report upon the brittleness of blades where the point is mounted, but space will not permit a discussion of the metallurgical problems concerned.

In spite of all that has been written, fencing remains an eminently safe sport, and one is far safer on the piste than on the road.

FENCING TERMS

1. PISTE: the area of floor upon which fencing takes place. Its width is 2 metres and its length 13 metres or more depending upon the weapon being fenced.
2. FOIL: this is a light weapon having a blade of rectangular cross-section. It is deemed to have a point only.
3. EPEE: this is the direct descendant of the duelling sword. It is heavier than the foil and has a metal coquille or hilt about 13 cms. in diameter and about 5 cms. in depth. It is deemed to have a point only.
4. SALLE: the room in which fencing takes place.
5. PRESIDENT: the official who controls the fencing and applies the somewhat complicated rules.
6. COQUILLE: *see under* EPEE.
7. SABRE: a light weapon deemed to have a point and cutting edge. The hilt or guard is shaped to protect the whole of the back of the hand.
8. POOL: a group of competitors who all fence each other successively.

10

GAME SHOOTING AND INJURY

J. R. ARMSTRONG
M.D., M.Ch., F.R.C.S.

MAN is carnivorous and must eat, so the pursuit of game in one way or another must have been one of his first activities. Originally a necessity, hunting quickly became a sport or pastime with a compelling lure which is no less today than it was at the dawn of history. Traditionally the British have always been field sport enthusiasts and shooting is second only to angling in popularity. There are few large wild animals in these small islands and the smoothbore gun, the fowling piece and its more modern counterpart the shotgun, and game birds or wildfowl are the main components of this sport.

ACCIDENTAL GUN-SHOT WOUNDS

Shooting should not be a dangerous pursuit but it is not without its hazards. Of these by far the greatest is the danger of the accidental shooting of one of the participants. The nature and treatment of gunshot wounds is outside the scope of this work. It should be the concern of all who use guns to prevent such accidents rather than to know how to deal with them when they do occur. Mr Joseph Nickerson, whose famous shoot at Rothwell has become legendary in his life-time, has outlined the basic safety precautions which I endorse whole-heartedly. As a surgeon, every season I am called on to deal with a number of patients who have sustained severe injuries as the result of shotgun wounds: and I am always appalled at the carelessness and sheer ignorance which are responsible for these accidents. A number are the result of irresponsibility. For the sum of ten shillings, payable at a Post Office, anyone can obtain a licence to carry a shotgun. This being so, it is unavoidable that these weapons should sometimes fall into the hands of people who know nothing about them and who do not appreciate or care how dangerous they are. Inevitably accidents follow, often involving innocent bystanders, and even strict legislation might not prevent this happening. More often, however, those responsible should and, in fact, do know better. Changed econo-

mic conditions have resulted in shooting becoming the sport of a far wider range of people than it was. Better transport and the establishment of so many 'syndicate' shoots have resulted in many townsmen taking up the sport, often for the first time in middle age. In general this is an excellent thing but it has its disadvantages. Unlike the countryman, who has been familiar with guns all his life and who has been grounded in the basic rules on which safety depends in boyhood and who has shot as a young man under the critical eyes of his elders, the townsman often has little real training at all. He does not appreciate that a shotgun is a lethal weapon, far more dangerous at close quarters than a rifle. Indeed his own efforts when the birds come over him may well lead him to suppose that it is comparatively harmless! A syndicate shoot has no host with powers to remonstrate, either tactfully or forcibly according to his nature, with guests if he considers them dangerous. The organiser is often in a difficult position when it comes to dealing with members whose success in their own walk of life may have rendered them intolerant of criticism, however tactfully expressed. Such people often remain quite unaware that they are dangerous. Although a careless shot may wound a beater or a fellow gun, in my experience most major injuries result from the accidental discharge of a gun which should be unloaded. At a large organised shoot, a number of people congregate together between drives; and if a gun goes off accidentally someone is bound to get hurt. To me the hallmark of a well-run shoot is the insistence that, at a signal marking the end of a drive, everyone unloads and carries his gun open until he receives the signal indicating the start of the next drive. Mr Nickerson's remarks about the peace of mind engendered by the open gun will be appreciated by everyone who, like me, has spent a miserable day as a guest at a strange shoot wondering how many of the guns conspicuously displayed between drives are actually empty. Moreover an open gun sets a good example to others. One of the best and safest shots I know, a man who visits a large number of different shoots each season, carries a large, slightly oily duster in his pocket which he wraps round the open breech of his gun when he has unloaded at the end of a drive and which he removes only to reload at the beginning of the next drive. Because of this practice his friends may pull his leg a little on dry days; but it is astonishing how many open guns are seen at any shoot at which he is present. He himself once confessed to me that he used his duster not so much to protect his gun as to protect himself from being shot 'by some damn fool with an empty gun'. Gunshot wounds are a vital concern of all who shoot, not as regards treatment but, what is better, prevention.

RECOIL INJURIES

When the trigger of a loaded shotgun is pulled, the hammer falls, driving the striker against the percussion cap of the cartridge. The friction produced explodes the contents of the cap and the resulting hot gases ignite the powder charge. The powder burns and is converted into gases which expand rapidly, generating great pressure in every direction. If powder were fired and confined in its own volume the pressure produced would be greater than any firearm could withstand: but in the shotgun the wads and charge are driven from the cartridge along the barrel. Provided the gases are released in this way, the walls of the chamber are sufficiently strong to stand up to the lateral pressure: and the main force of expansion is directed forwards and backwards, these forces being equal. The amount of force generated depends on the resistance to expansion encountered, and this resistance is determined by the weight of the shot charge and wads. The forward force propels the shot towards the target, the backward force, falling on the face of the action, drives the whole gun backward, producing the so-called recoil or 'kick'. The force of recoil is measured by the momentum imparted to the whole gun, and momentum is measured in terms of mass multiplied by velocity. The mass is the weight of the gun and the velocity can be measured in feet per second. The velocity of recoil in fact principally determines the effects; and long experience has shown that a velocity of 16 ft. per second is the most that can be withstood with any comfort, with some individual variation. When a twelve bore shotgun weighing six-and-a-half pounds is fired with an ordinary two-and-a-half inch cartridge loaded with one and one-sixteenth ounce of shot the velocity of recoil is 15·4 ft. per second. With a six and three-quarter pound gun it is 14·8 ft. per second and with a six-and-a-quarter pound gun it reaches 16 ft. per second. When a one ounce shot load, which reduces the resistance to expansion and, therefore, the pressure generated, is used there is a decrease of about 5 per cent in the velocity of recoil.

When a shotgun is mounted the stock rests against the shoulder, where it should bed down firmly in the groove on the medial side of the deltoid. When the gun is fired the main force of recoil is taken on the shoulder itself, although the arms help by resisting and damping down the 'kick' to some extent. Although most experienced shots are quite unaware of and unaffected by recoil this force may, in some circumstances, cause various injuries and ill-effects.

Bruising of the index or middle finger by the trigger guard is sometimes troublesome: and I have seen a fracture of the proximal phalanx caused in this way. Normally the triggers are pulled with the index finger while the stock is gripped with the rest of the hand, the second finger lying just behind

and clear of the trigger guard. If the stock is ill-fitting or badly shaped, the guard may impinge on this finger each time the gun is fired. Attention to stock fitting, alterations in the shape of the trigger guard or padding of the guard with sorbo cures this complaint.

A particularly unpleasant recoil injury may happen to the right thumb. Many people push the safety catch off as they mount the gun, thus bringing the tip of the thumb close to the end of the lever. If the gun is fired with the thumb in this position, recoil drives the lever backwards against the extended thumb producing a vertical splitting which often involves the nail and nail bed.

Bruising of the shoulder is quite common; and, if severe, it is always due to an ill-fitting stock or incorrect mounting.

When a gun is properly mounted, the right cheek is in contact with the stock and some people suffer bruising of the face. Severe bruising is not uncommon in those who suffer from 'gun headache' and is probably due to the same physical peculiarity. Bruising of the cheek is easily prevented by the fitting of a suitable pad to the stock.

Gun headache, the severe migrainous type of headache which affects many people in the course of a day's shooting, is a most unpleasant affliction. There has been considerable argument about the aetiology and many 'cures' have been advocated. There can be little doubt that the true gun headache is of cerebral origin, being produced by the repeated jarring or jolting of the head by recoil. Exactly the same type of headache occurs in boxers as the result of too many blows on the chin or head. The cure is to reduce the velocity of recoil to within the tolerance of the individual; but some people find that even the lightest possible recoil causes headaches from time to time. In my experience many of those troubled in this way have lesions of the cervical spine, usually osteoarthritis, which have reduced the normal mobility of the neck. In such people, recoil is transmitted directly to the head without being buffered by neck movement, and the ill-effects are consequently increased.

Occasionally more severe injuries occur. Fracture of the coracoid is not excessively rare, and brachial plexus palsies of various degree have been reported. Some of these injuries are the results of freak accidents. From time to time people are sufficiently ill-advised as to discharge a shotgun when they are not in a position to move with the recoil. If someone who is leaning against a tree or a wall, for example, fires a gun the shoulder cannot 'give' freely and some injury is bound to occur.

The basic cure of repeated recoil injuries lies in the hands of the gunfitter or gunsmith. The first step is to make sure that the stock is the correct length so that it beds firmly against the shoulder when the gun is mounted. The

effects of recoil may be reduced by using a lighter shot load (one ounce being usual) or a heavier (not a lighter as so many people suppose) gun. Most gunsmiths market a light load cartridge which is suitable for those who suffer from recoil phenomena.

'BURST' INJURIES

Fortunately the bursting of a shotgun barrel is a rare occurrence. From time to time these unpleasant accidents do happen, however, for one of three reasons. The barrels themselves may be defective as the result of neglect, years of use or an unsuspected flaw in the steel; and many burst under the pressure generated by a normal faultless cartridge. Occasionally a cartridge may be faulty and may generate an abnormally high pressure which bursts a sound barrel: but for practical purposes such occurrences are only possible if hand-loaded or cheap foreign cartridges are used. The third, and by far the most common cause is obstruction in the barrel when the gun is fired. Obstruction may be of various kinds, but mud or snow, a piece of cleaning material, a twenty bore cartridge loaded in mistake for a twelve or a shot charge and wads not ejected by a defective cartridge are probably the most common.

When a barrel bursts, recoil is enormously magnified; and some injury is almost inevitable. In addition, damage to the barrel itself may involve the left hand. The appearance of a burst gun is so alarming that one might imagine that serious injury to the person firing it was inevitable. However, Major Sir Gerald Burrard, the foremost authority on the shotgun in Britain, states that although he has seen some hundreds of burst guns he only knows of serious injury on four occasions. In the only instance of which I have first hand knowledge, a first-class gun in immaculate condition burst in the course of a day's shooting because of an unsuspected flaw in the barrel. The owner was knocked unconscious and for some time had a median paralysis of the left hand but sustained no permanent injury.

With the exception of the unsuspected flaw, all bursts occur as the result of some degree of carelessness and ignorance on the part of somebody, and are therefore avoidable. A gun with such a flaw may pass proof as this is carried out today, and indeed may last for some years until the steel becomes 'tired' at the weak point, when it will burst. There are, however, modern methods which will detect such hidden flaws: and in spite of the cost involved such tests will sooner or later become part of the normal proofing procedure.

SUMMARY

Many shooting injuries are due to carelessness and ignorance and the rest are matters for the gunsmith or gunfitter. As with most other things connected with sport, the only wise course when trouble arises is to seek first-class professional advice rather than be guided by well-meaning counsel from amateurs.

GAME SHOOTING AND INJURY

JOSEPH NICKERSON

ACCIDENTS occurring in the shooting field are almost without exception attributable to carelessness and there are some things about which it is impossible to be too particular.

Obviously it is absolutely essential to use only a weapon which has been tested by the Government Proof House and bears the officially correct proof marks. Many guns in use today are getting very ancient and in the interests of the owners should be returned to the Proof House to make sure that they are safe. In my career I have seen several guns whose barrels are worn almost to the thickness of paper and have handled several that have actually had holes in the side of the barrels. These are often treasured possessions of their owners, who are loath to part with them and are therefore afraid to have them re-proofed, knowing they may fail to pass the test.

Shooting often takes place in wet weather and on ploughed land, and it is therefore easy for mud to lodge in the barrels, causing them to burst. This would not hurt the actual person firing the gun if the blockage were at the muzzle; but anyone standing to one side of him, for example his neighbouring gun, would be in great danger. Particular care must be taken in snowy weather for an obstruction of snow almost always causes a burst barrel. My loader always carries a pull-through with him, and on the mud of the estuary or in snowy conditions this has proved very useful, preventing the loss of much time.

Shotguns should be carried open, that is with the breach broken. Not only is this a simple safety precaution which I insist on at all shoots under my control, but it also gives peace of mind to the whole party who are visibly aware that the gun is not loaded. Whenever a gun is handed to you, you should make it a 'drill' to open it to see if it is loaded. I have done this all my life, irrespective

FIG. 1. The gun swings round to take a bird behind while his loader and spectators get well out of the line of fire.

of the importance of the person who hands me the gun, and unless anyone does likewise when I hand him mine, I mark him down as a source of potential danger.

When a gun is closed the stock should be brought up to the barrels and not the barrels to the stock; if the latter is done the barrels point towards body height, whereas if the former is done they point towards the ground.

My shooting coats are made without buttons on the cuffs and with only one button on the front of the jacket, which should always be fastened.

The safety catch should only be pushed forward as the gun is mounted to take a shot. When birds are passing through the line one often sees people 'swing through the line' instead of dropping the gun and remounting to fire a shot behind. Ricochet from stones, tree branches, or the toe or beak of a bird are far more common than most people realise, and can strike hard enough to blind at a phenomenal distance. Do not shoot where you cannot see. If some of the guns were to walk in the beating line occasionally they would realise how the beaters feel when, towards the end of a drive, a low bird comes forward. This is particularly true in hilly country where a shot at

a bird of even moderate height could be a great danger to the beating line. I always instruct the beaters before going over a crest to announce their approach audibly. The man on the outside of each end of the line of beaters should carry a red flag so that the guns know he is the last in line. Believe it or not, I have seen some lines of beaters where the outside pairs of men, that is four in all, have red flags.

When waiting for driven game, hold your gun upwards or pointing towards the ground. If you have a retriever standing in front of you, do not point your gun towards it during a drive. I have seen a strong badly-broken dog tied to a shooting stick with a man sitting on the shooting stick holding a gun in his hands. I have also seen him deposited on the ground on his rear on the arrival of a hare in the neighbourhood.

It is very important that the loader should be properly drilled; and if one does not know how to train him one should not be too proud to send him to a shooting school. An accidental shot by a loader would almost certainly be fatal to his employer. When you use a pair of guns and a loader, having discharged one barrel always slip the safety catch into the 'on' safe position before handing it to your man.

Before loading your gun in the morning always look through your barrels and instruct your loader to do likewise. Make sure that your loader holds the second gun pointing to the sky and that when he loads it he always turns away from you to do so. He, too, must bring the stock up to the barrels and not the barrels up to the stock.

Never leave a gun standing on a concrete pathway or road as it could be knocked over by dogs and severely damaged, and might afterwards be unsafe. Guns in vehicles like Jeeps or Land Rovers should be carried in special gun racks, pointing not sideways but upwards through the roof. The clips in front of these racks should carry a safety chain as well; for when sitting in a vehicle travelling on bumpy ground I have seen a gun come backwards, hitting the passenger at about eye level and this I can say from experience is both irritating and painful.

When hands are cold it is possible for the shooter to discharge the gun as he mounts it, since the fingers are numb and have not the natural feel of the trigger. One shoots better when the wrists are warm, so that gloves with a woollen wrist are helpful. Personally I wear some very fine gloves, which can be obtained from most gunmakers now; and although at first I was loath to wear them, I have found them a tremendous boon. If in a warm corner, one has to fire a dozen shots as quickly as one can load, the metal of the barrel becomes too hot to hold and the gloves protect you from heat as well as cold. In very wet weather, however, these leather gloves can be dangerous as they

are very slippery. For these conditions one should carry a pair of woollen gloves and the ones I have are mittens, the wool only extending as far as the knuckles.

Gun headaches are often caused by firing too heavy a load for the weight of the weapon. The lighter the weapon the lighter should be the cartridge load. From a 7 lb., 12 bore gun one could fire $1\frac{1}{4}$ oz. of shot without getting a headache, but from a 6 lb. gun even the toughest of us would be uncomfortable with more than 1 oz., even if the gun were proofed for this weight of shot. Cartridges should be stored about the temperature necessary to keep leather bound books in good condition. I have known people put damp cartridges in a very high temperature; but this affects the powder and will produce gun headache in record time.

If the gun does not fit the shooter, that is if the length of stock is not correct, a bruised finger can be the source of great pain, leading to inaccuracy. A visit to a shooting school can remedy this, but a small rubber pad fitted behind the trigger guard also helps. I like the first trigger of a double trigger hinged so that it gives against the trigger finger when one pulls the second trigger.

If the gun is not correctly mounted to the shoulder, naturally the recoil causes the butt to strike the body, bruising the shoulder. This sometimes happens with a very quick shot. It is always essential to mount the gun correctly and place it firmly into the shoulder.

11

GOLF

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MAN'S BODY image is built up from the complex flow of nervous impulses coming in from proprioceptive sense organs throughout his tissues and his delicately adjusted balance mechanisms. He extends this body image to incorporate the tools he uses and the implements he adapts to satisfy his sporting needs. Thus his body image may be required to extend to the front and back bumpers of a car, to the tip of an electric drill, or the head of a golf club. The expert player of ball games has a highly developed body image. He has also the delicacy of control and co-ordination necessary to give perfect judgement of force, and exact timing. This absolute accuracy is required to strike the ball with the exact impact and in the exact direction which is necessary to place it where the player judges it should land. The game of golf is an extremely searching test of this type of skill.

The ball and club head are of relatively small size. Elasticity of the ball, whip and torque of the club shaft and elastic stretching of body tissues under the influence of the tremendous centrifugal force used in the full golf swing, lead to a great number of possible errors in bringing the club face accurately and precisely against the ball. This concerted effort at powerful propulsion must be contrasted with the slight movement and delicate control of power needed for the short game. It is only the exceptional performer who can achieve proficiency in these varied skills. There is so little margin for error that correction by proprioceptive control is frequently required during the very process of the swing.

The amount of strain placed upon the body tissues when striking the ball varies considerably from that of the ungainly dipping heave of the clumsy 'rabbit' to the smooth flow and pivot of the expert professional. Almost every muscle in the body is involved at one time or another from the intrinsic muscles of the feet to the sternomastoids and the strains placed on the components of the locomotor system are severe. Tournament golf is no Sunday

morning stroll, as anyone will know who has played seventy-two holes in a half gale on an exposed coastal course. No other game requires its accurate shots to be played on such varied terrain and at such awkward heights and angles. No other game involves the long periods of inactivity which allow cooling off and stiffening between the shots. Mental stress is added to physical strain by the sustained psychological tension and the war of nerves imposed by tournament play. Individual games require a different psychology from team games and the self-reliance to stand alone upon the stage—often before a large audience.

Whatever method the golfer uses to propel that small white ball along the course, experience shows that the strain falls greatest upon his back. Strains of the lumbar joints and less frequently those of the cervical and thoracic

FIG. 1. *Left*: even a first class professional sometimes seems to make an all-out effort when he hits a golf ball: *Right*: perfect poise and control shows the complete absence of physical strain when executing a shot.

spine are by far the most common injuries encountered. Here, posture, skill and ability, together with general physical build and proportion of limbs to trunk, play their part. Minor congenital abnormalities from half an inch difference in the length of the legs, to transitional or abnormally shaped vertebrae, may predispose to injury. Local muscle strains are far less common than joint derangements, where the pathology may vary from minor ligamentous strain to a full prolapse of an intervertebral disc with nerve root pressure. The presence of relatively stiff areas in the spine from old osteochondritis, or the early wear-and-tear changes of spondylosis, are frequent predisposing factors. Strain usually occurs where a mobile joint adjoins a stiff one. There are also more subtle influences such as the splinting action of the diaphragm, extending down to the third lumbar vertebra, if the breath is held in an attempt to give added power to the shot.

In the older spine degenerative changes of spondylosis causes narrowing of neuroforaminae and mushroom bulging of discs, with increased liability to root pressure. Relative shortening of the spinal column in relation to the spinal cord alters the angle of pull on the lower cervical and upper thoracic nerve roots, giving an added factor to interfere with local vascular supply and cause increased liability to nerve root oedema and pressure.

Moving outward from the spine, shoulder lesions are relatively uncommon. This is probably because these joints are not taken through the range of movements above the horizontal, so that the muscles of the rotator cuff do not act at a mechanical disadvantage. Greater strain is taken on the wrists and forearms in controlling and adjusting the swing at the moment of impact and a fairly frequent lesion is 'tennis elbow' or strain of the extensor muscles at their origin from the lateral epicondyle of the humerus. Strain at the origin of the flexor muscles from the medial epicondyle is much more rare, although it bears the name of 'golfers elbow'. The hips seldom suffer in golf unless they are the site of abnormality or osteo-arthritic change.

Considerable lateral and rotation strain may be placed on the knee joints, especially when playing from an awkward lie with the feet at different levels. Strains of the medial ligament and the occasional cartilage lesion are therefore seen, usually in those over the age of thirty five. Ankle strains are rare in actual play, but the hazards of rough terrain and the careless placing of a tired foot wearing shoes designed to grip the turf, may lead to local sprains.

Most of the tendon lesions such as tennis elbow, medial ligament strain at the knee, lateral ligament strain at the ankle and the occasional occurrence of painful heel in the middle-aged or elderly are due to underlying degenerative changes in the fibrous tissue structures concerned, with the super-

imposed trauma of the game acting as the trigger which starts off inflammation.

Injuries due to direct contact with ball and club are unusual, but it should be remembered the golf ball, like the squash ball, is small enough to enter the orbit and cause severe damage to an eye. When travelling fast it is quite hard enough to cause severe contusion of soft tissues, subperiosteal haematomata and even local cracking of subcutaneous bone.

As in all games, adequate training, proficiency, correct posture and perfect balance minimise physical strain. What advice can therefore be given to golfers young and old, professional and amateur, to guard them from the hazards of their tantalising but delightful sport? For the professional or amateur engaged in tournament play it is important that adequate general physical training should be undertaken before the season starts. This is best carried out in a gymnasium, ideally under instruction from a qualified gymnast, with facilities for full circuit training. Road, or cross-country running and walking are best for building stamina and endurance. Specific weight lifting can be used to balance development and counteract individual muscle weakness. The hazards of weather and terrain cannot be altered by the player; he must guard against them by careful selection of clothing, including footwear. Lightness and looseness allowing freedom of movement must be combined with adequate protection against chilling during the periods of inactivity. Loosening exercises and practice play should be carried out before each round and every chance should be taken to relax muscles and nerves between shots. Medical advice and physiotherapy treatment should be sought early if painful strains result. If one structure or part of the body gives recurrent trouble, remedial exercises, strict attention to posture and even minor modifications of style may be necessary to prevent serious injury.

Apart from the game itself, certain commonsense precautions are advisable. Because of the importance of back strain, unnecessary heavy lifting and awkward bending strains should always be avoided. Good posture should be sedulously cultivated and for those who travel long distances this is specially important. Lack of attention to a sagging bed or the height of pillows when the body is tired may lead to joint strain during sleep. On aircraft and motor transport, it is worth using a lumbar support. A rolled rug or small pillow should be placed in the small of the back during periods of fatigue on all long journeys. A simple exercise programme should be regularly carried out. For the amateur the same advice applies in a modified form. He must remember that pains or aches coming on within a few days or even a week of play may be due to the game and careful thought may be necessary to determine whether other activities have placed a strain on the part of the body involved.

In conclusion, it should be remembered that a high neurotic score is found among almost half of all patients complaining of painful conditions. The game of golf places a considerable mental strain upon its exponents and devotees. Adequate explanation of any lesion and reassurance on the part of the medical attendant or physiotherapist is essential to gain the confidence and co-operation of those under treatment for this type of athletic injury. Most athletes have a strong will to get well and have a great fear of disability. Time spent on full explanation and discussion of their injuries is of the greatest value to them.

GOLF

TOM HALIBURTON

British Ryder Cup Team

TOM HALIBURTON, who still holds the record for the lowest score on a British course, confines himself neither to tournament play nor to teaching; but whether he is playing with his members, practising, giving a lesson or competing he spends a prodigious number of hours on the golf course, and is familiar with the various disabilities which may occur.

We found him out on the course at Wentworth and asked him for his views.

'Large handicap players don't seem to get any special troubles' he said. 'They seldom swing in exactly the same way in consecutive shots—this is the chief reason they never get their handicaps down to single figures. They are not much given to practice either, and a round of golf has more walking than hitting in it. For these two reasons they are not likely to over-exercise any particular group of muscles. The expert, on the other hand, often gets muscle trouble, expecially in the small of the back. This is the part of the body which does a lot of work in a golf swing, and the expert uses these muscles in the same way time after time. The real problem is that he spends far more time practising than playing. He stands in one spot with a large bag of balls hitting them one after the other in much the same way.

'Another result of all this concentration practice is characteristic "stance posture", so that even at rest the professional often looks as though he is getting ready to hit a drive. It is a shoulder stoop, almost a deformity—the left shoulder up, the right shoulder down. You can pick out some professionals on the course miles away. I have to have my own jackets cut to disguise the way my shoulder blades stick out—you could lay a tea tray across the top of

my shoulders. I tell my assistants to give as much attention to keeping their shoulders normal by doing remedial exercises as to practising golf shots'.

He showed us his hands which are covered with large callouses. 'Most golfers have these' he said, 'but only the experts have really noticeable ones—mostly on the thumb and on the heel of the left hand, and the index finger and ring fingers of the right hand. Since I started wearing a glove they seem to have improved.'

We discussed 'golf elbow' with him. It is not as well known as 'tennis elbow', yet it is a very similar condition—indeed the site and the lesion are identical: but 'tennis elbow' which, when it was originally described, used mainly to effect the little finger side of the elbow (the medial epicondyle) now, because of the difference in the technique of shot making, effects the thumb (or lateral epicondyle) side. 'Golf elbow' is now the common term for a lesion affecting the medial epicondyle. Tom Haliburton thinks that the condition begins with some unexpected jolt, as when a player hits hard into deep rough and the club head suddenly stops. The deceleration may be considerable so that the hands are torn from the grip.

Most of the leading golfers, he told us, particularly those whose livelihood depends on the game, are careful to warm up before hitting full out from the first tee. This is most important in cold weather; and the various tricks adopted by the professionals are not reminders of technique (as the practice swings of the week-end golfers usually are) but muscular preparation and inducers of co-ordination.

As we thanked him and walked away, he went back to his practice, hitting ball after ball in the same way to the same place.

12

INJURIES IN HOCKEY

NORMAN BORRETT

Captain, English Hockey Team
Captain, English Olympic Hockey Team

HOCKEY, unlike the two other major British winter games, seldom has injuries brought about by the personal contact between players, because it is against the rules for a player to use his body to check an opponent.

This does not mean that collisions do not occur. It can be an extremely fast game, and when it is closely contested there are certain to be occasions when two players knock into each other. Determination and dash are bound to produce cracked heads and body bruises, and no game worth its salt is free from danger of this kind.

Most hockey injuries are brought about by the ball, or the stick, and these injuries vary with the state of the ground and the proficiency of the players. A billiard table surface should reduce injuries to a minimum, but this perfect surface is the exception rather than the rule in the wet winters of the British Isles. Muddy, bumpy and frozen grounds cause the ball to fly or rise, and then the players are more likely to get bruised legs and bodies and possibly cut faces. The last is fortunately infrequent, but does happen. The same injuries may be caused by players flicking or hitting the ball into the air, whether legally or illegally. The ball may also shoot up and hit a player's face when two players go for it at the same moment. The sticks converge from behind the ball on opposite sides of it, and up it flies. One unlucky international lost an eye in this way; but the bones around the eye normally guard the eye from the danger of a ball as large as a hockey ball.

The stick, like the ball, when used carelessly or ruthlessly, causes similar injuries. These may be bad bruises or cuts on the legs or hands, and in some cases, again fortunately infrequent, on the face. I have seen one or two nasty accidents to players' mouths, e.g. a stick raised too high on the backlift removing a player's front teeth. Sometimes when a player tackles on the wrong side he is inclined to bend his body so that his head is only waist high. If his opponent hits the ball and follows through with his stick, he is quite likely to hit this player on the head.

The only safeguard against cuts and bruises on the lower leg from the ball

FIG. 1. First class hockey played at top speed must inevitably carry some risk of accidental injury.

and stick is the wearing of light shin-guards, which take some of the sting out of a hard blow. It should not be thought 'soft' to wear shin-guards, because if a player receives a hard blow and is not wearing some protection he may finish the match off the field—much to his own and his team's chagrin. Remember that a hockey ball is hard and can be hit as hard as a cricket ball. No one can protect himself above the calf; quickness and a knowledge of what might happen under various circumstances are the greatest help here. Most players learn to take these knocks even though the hard ones can be extremely painful. Personally I always played when badly bruised because I enjoyed the game so much and was prepared to put up with the pain for the fun of playing. During a game, after a hard blow, it always seemed better to continue playing, for that took one's mind off the bruise.

Another injury common to most active games today is a pulled muscle. I cannot remember this being nearly so prevalent before the last war as it has become since. Some players seem to be addicted to muscle trouble whilst others never suffer from it. I cannot recall ever having pulled a muscle playing hockey, and I feel that to avoid this injury everyone would be well advised to get well warmed up before starting a match by running round the ground.

My own injuries have fortunately not been serious and I doubt whether

many hockey players have had a different experience. I have had a number of near misses when the ball has rocketed by my face, and have stopped my fair share of such hits on the foot, legs and body. I have also had some painful cracks on the foot from opponents' sticks and must have had a fair area of skin removed from my fingers: but although uncomfortable at the time the lasting effect is short. Once I was kicked very hard on the thigh by a goalkeeper, and that produced, so my doctor told me, internal bleeding; but this did not stop me playing three days later even though I was strapped up. Few players I should imagine have been felled by a goalkeeper in this way although many forwards may have collided with their 'opponents' goalkeeper. This may cause a broken rib or concussion if the impact is hard enough.

It was at the end of my international career that I developed osteo-arthritis, and I can hardly lay the blame on hockey as I played other vigorous games as well and for most of the year. This started in my right hip, and I often wonder whether this may have been caused partly by my being an inside-left and having to swivel frequently on my right hip to hit the ball to the right. However, I know of many inside-lefts who have played the game for years and are not troubled by their hips.

Cartilage trouble also bothers some hockey players, but I must admit that this does not seem to be very common in this game. Some sufferers have come into the game after playing rugby football, in which game they first had cartilage trouble, so that hockey can hardly be blamed. There is a lot of quick movement, stopping, and twisting in the game and it would appear that sudden turns can cause trouble, especially to the knees. Happily such occurrences are uncommon, and are more likely to happen when playing on a frozen surface.

Lastly there are a number of injuries which really are rare. Three come to mind, a broken collar bone, Achilles tendon trouble and concussion. The only case I know of a broken collar bone was caused by two players colliding. The Achilles tendon trouble I believe started in a rackets' court; and the concussion was caused by a back falling over just as a forward hit the ball with all his might. The back's head stopped the ball, and although he played on for some time he had to be led off eventually. Recently I saw a goalkeeper concussed after a heavy collision with a hard running forward when they were both trying to get to the ball first.

It is a great recommendation for hockey that players continue performing actively into their forties, provided, of course, they keep fit; and it is not unusual to find internationals still playing for their counties in their thirties. Naturally this long playing period is due in large measure to the absence of body tackling.

The most common injuries are therefore bruises and cuts—the general wear and tear of the game. Pulled muscles and cartilage trouble are much less frequent, and other injuries might be classed as unusual, rare and 'out of the blue'.

13

RIDING ACCIDENTS

R. P. GOULDEN
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FROM the records of a London clinic, two hundred and eight injuries sustained by fifty-seven steeplechase jockeys over a ten year period have been analysed. From this analysis it was hoped that a pattern would emerge indicating how these injuries occurred, which injuries were the most common, and if possible to suggest measures which could be introduced to reduce the accident rate.

The exact mechanism of injury is frequently very difficult to determine as it happens so quickly. In addition, after the rider has fallen, he is subjected to the terrifying experience of lying on the ground with several horses galloping over him, frequently kicking him, and it is small wonder that seldom can he give any account at all as to how the accident occurred. An additional hazard is that of the horse rolling on the rider. Rarely is an eye witness available, so that slow cinematography is the only method of solving the problem exactly, although a rough idea can usually be obtained from the injuries themselves.

Some jockeys are more prone to injury than others. One sustained twenty injuries, one fifteen, one fourteen and another thirteen, some of them major injuries. These four jockeys frequently sustained more than one injury at a fall, and were usually kicked after they had fallen. They are all completely fearless and possibly took more risks than the others. They were also probably leading the field and therefore stood a greater chance of being kicked by the following horses. They also had many more rides in the season.

It must be emphasised that the analysis is of a selected group of jockeys, those whose injuries were not so severe that they had to be admitted as emergencies to hospitals near the race courses. Though the exact number of these severe injuries in the time is not known, nor the number of jockeys treated at other similar metropolitan clinics, the broad pattern of injury is probably clear enough.

Several interesting points arise from a study of these riding injuries. Not a single patient developed an accident neurosis: their first and only thought was to return to racing again as soon as possible. They are a very refreshing

TABLE OF INJURIES

1. Fractured clavicle 40
2. Severe bruising without bone injury 33
3. Acute sprain of cervical spinal joints 19
4. Dislocated shoulder (4 recurrent) 11
5. Subluxation acromioclavicular joint 10
6. Concussion 10
7. Knee joint:— 10
   (a) comminuted fracture of patella 1
   (b) torn medial meniscus 1
   (c) Traumatic effusion 3
   (d) Partial tear medial ligament 3
   (e) Haemarthrosis 2
8. Multiple fractured ribs 9
   (simple) 7
   Multiple fracture ribs (with pneumothorax) 2
9. Fractured metacarpals 9
10. Crushed fracture. Dorsal vertebrae 6
11. Fractured ankle 7
   (a) Medial malleolus 5
   (b) External malleolus 1
   (c) Tri-malleolar 1
12. Fractured bones in foot 5
13. Fractured femur 4
14. Fractured fibula 4
15. Sprained metacarpophalangeal joint of thumb 3
16. Fractured scaphoid 2
17. Fractured transverse process lumbar vertebrae 2
18. Avulsion greater tuberosity of humerus 2
19. Fractured humerus with radial nerve palsy 1
20. Fractured lamina with subluxation C2 on C3 1
21. Fractured base of skull 1
22. Dislocation sterno-clavicular joint 1
23. Fracture medial epicondyle of humerus 1
24. Compound fracture dislocation shoulder 1
25. Impacted fracture neck of humerus 1
26. Fractured neck of radius 1
27. Fractured ulna 1
28. Fractured radius and ulna 1
29. Fractured proximal phalanx of thumb 1
30. Fractured semilunar 1
31. Fractured tibia 1
32. Severe laceration of calf muscles 1
33. Strain of adductor longus 1

section of the community to treat. All of them returned to racing, including one who was advised to retire after three bad concussions. Even though his electroencephalogram was normal it was felt that his judgment was impaired, and that he was becoming 'accident-prone'.

The most common cause of injury is when the horse stumbles or falls on landing from a jump, the rider being pitched forwards. This accounts for the

FIG. 1. Failure on the part of horse or rider can result in this.

fact that 85 per cent of these injuries are situated above the waist, as it is the upper part of the body which hits the ground first. The most frequent site of first impact is the shoulder: thus 20 per cent of these injuries are fractures of the clavicle, 5 per cent subluxation of the acromio-clavicular joint and 5 per cent dislocation of the shoulder. A fall on the head or upper dorsal region, causing acute flexion and hence severe sprain of the cervical spine accounts for 10 per cent of these injuries: and yet there was only one broken neck—a fracture of the cervical spine with subluxation of C2 on C3. Intervertebral disc degeneration with osteophyte formation, however, is common at an early age, appearing as a complication of these injuries. However, cervical spondylosis seems to cause remarkably little disability, even in later years. Concussion (accounting for 5 per cent of the injuries) was frequently due to kicks from following horses. Only one fracture of the skull was encountered.

Fractured ribs and pelvis were due to direct kicks and to the horse rolling on to the rider.

The trimalleolar fracture was caused by a stirrup leather breaking.

The severe laceration of the muscles and tendons of the calf was due to a sharp projection from the rails ripping the rider's legs as he passed at speed.

Another interesting point was that only one peripheral nerve injury was seen; this was a complete radial nerve palsy associated with a fractured humerus which took six months to recover sufficiently for the patient to race again.

PREVENTION OF RIDING ACCIDENTS

Steeplechasing always has been and always will be a dangerous sport, and jockeys will get hurt. There are so many variable factors which contribute to these accidents and so few which can be controlled. In particular, form, whether of horse or rider, is unpredictable; and weather is beyond human management. But there are measures which can be taken.

The rails and jumps, for example, should be examined for projections. One jockey sustained severe laceration of the muscles and tendons of his calf and a compound comminuted fracture of his fibula, when he caught his leg on a projection from the rails. This could have been prevented.

Harness and equipment should, of course, be absolutely first class. One trimalleolar fracture dislocation of the ankle was caused by the breaking of a stirrup leather.

Jockeys must be fit, and must not be allowed to return to racing too soon after an injury. This applies particularly to the young, relatively inexperienced riders, who are trying to make a name for themselves, and to whom the riding fee is all important.

Protection to the backs of the hands might reduce the risk of fracture of the metacarpals, of which there were 9 cases in the series.

It is tempting to think that these accidents could be reduced by teaching riders how to fall correctly, as are trainees in the art of Judo. Some riders take bad tosses and get away without serious injury more frequently than others, and they seem to know instinctively how to fall.

It is sometimes suggested that only well trained and experienced horses should be allowed to race, an idea unlikely to commend itself to anyone since, if novice chases were eliminated, there would be nowhere for a young horse to get race experience. Injuries are less common, also, when the ground is soft: but meetings are not likely to be postponed because the going is hard, except in extreme conditions.

Experiments are being made at the moment with new types of helmets in order to give the rider greater protection without increasing his weight. This is very important and will no doubt reduce the number of cases of concussion.

Steeplechasing is bound to remain a dangerous sport however hard one may try to reduce the accident rate. It is not generally realised that on an average three steeplechase jockeys are killed every year. The riders know the risks and accept them; all they ask is that they should get prompt and expert treatment when they do get hurt, so that they can return to the sport they love as soon as possible.

RIDING

PAT SMYTHE

International Horse Riding Champion

SERIOUS accidents are rare in riding, but stiffness and bruises are accepted even by the most experienced horsemen as an inescapable part of the sport. Pat Smythe, during her remarkable career, has had her share of accidents, some serious, many trivial: she has also managed to avoid injury in falls because she understands how injuries are produced: and she has given us the benefit of her insight.

Direct injuries are nearly always the result of a fall of horse and rider, and a high proportion of these falls occur during jumping. When the rider loses his position in the saddle, he may strike the horse's neck with his face, damaging his nose or loosening a few teeth. The same impact nearly always dislodges the hard hat (which is intended to protect his head), partly because the hat has a hard peak which acts as a lever and partly because it is unfashionable to wear the chinstrap, which is designed to keep the hat in position. Convention is, in this instance, not on the side of safety, which would be better served by a soft peak and the proper use of the chin strap.

If the horse and rider fall together, there are a number of dangerous positions. In a fast fall, the rider is usually thrown clear and his main danger is impact against some hard object—a tree, a fence or a wall. This is only likely to have really serious consequences if the impact is upon the unprotected head; but fractured collarbones, or bruised or broken ribs are usually the extent of the damage. It is the slow fall which produces the worst, sometimes fatal injuries, because the rider never really becomes separated from the animal, which may roll over him or, in struggling to rise, strike him with its hooves. If the rider has lost his hat and his head is struck by a shoe in which studs have been screwed for show jumping, a most severe injury results. One prominent rider was killed at Dublin in this way. Pat Smythe received

a most unpleasant muscle wound in the leg from a similar accident. These are the falls in which legs are broken and ribs crushed. Compression fractures of the spine sometimes occur, both in the neck and the back.

Indirect injuries are no less uncommon, the most frequent being low back strain from a twist during a fall. According to Miss Smythe, expert riders are more prone to stiffness than the beginner if quite a short times passes without a ride, presumably because they really work the horse rather than sit passively on his back. A horse which pulls hard may cause strain of the arms and shoulder muscles. Beginners who ride in trousers are bound to receive severe chafing of the skin of the calves and knees.

Miss Smythe considers that the rider can best prevent serious injury by falling away from the horse, releasing the reins at once so as to effect clean separation, and rolling away as soon as he hits the ground. Only if he has the ill luck to meet some unresisting object is he likely to be injured by impact: but against this possibility he should wear a hard hat which is properly designed, properly secured and will stay on during the fall. In addition to this specific advice, she is convinced of the value of physical and mental alertness which can so often prevent an incident from becoming an accident, or an accident from becoming a tragedy.

14

TENNIS ELBOW

M. SLAPAK
M.A., M.B., B.Ch., F.R.C.S.
Cambridge University Tennis Team

THE advantages of an experienced layman's point of view on an athletic injury emerge very clearly in Mr. Maskell's article. The qualities which have made him a world famous coach are underlined in the acuteness and accuracy of his observations and the shrewdness of the deductions he draws.

Tennis elbow is a blunderbuss term, often used wrongly to include all the pains affecting a tennis player's arm. The truth is that not every ache in a tennis player's arm is tennis elbow and conversely less than half of those who suffer from the complaint have ever played tennis.

The injury occurs in badminton and squash players, in golfers, miners, fencers, oarsmen and even violin players. Certainly the most classical case I have ever seen was in a lady, whose nightly task it was to appear at a West End theatre waving a sabre, as she performed an exotic dance. Mr. Maskell's own 'elbow' was triggered by a sudden rotary movement on the steering wheel of a car.

Tennis elbow is best defined as an injury which shows itself by a pain of varying severity more or less localised to a bony point situated on the outer or inner side of the elbow. The pain may spread a little way along the muscles of the forearm, but is maximal when pressure is exerted on the bony prominences. The injury on the inner side is often called 'golfers' elbow but frequently occurs in tennis-players.

Pain on the outer side of the elbow is accentuated by shots on the backhand side and the 'kick' service. Sliced strokes, particularly the wide, low, backhand volley tend to be specially painful. The basic pain producing movement is a straightening of the elbow while the forearm rotates away from the body. This is the commonest type of tennis elbow. Pain on the inner side of the elbow is associated mainly with the fore-hand drive (particularly if top spin is used), the service and the smash. Here the common movement is a bending of the elbow at the same time as the fore-hand rotates towards the body.

It seems reasonable to accept that the actual injury which causes tennis

elbow is probably a partial tear of some of the muscle fibres attached to the inner or outer side of the elbow, and that this tear occurs in association with certain movements of the forearm. The evidence of this belief is, however, indirect. Few sufferers are sufficiently desperate to allow operation on their elbow. Few doctors, fortunately, believe that an operation in order to confirm the presence of torn muscle fibres is justifiable, and to produce tennis elbow experimentally in, say, a guinea pig is an understandably difficult procedure.

The tearing of muscle fibres releases certain damaging chemical substances in the vicinity; and their presence causes the pain and the swelling around the bony prominences of the elbow. Sometimes the overlying skin becomes a little reddened.

But why does it all happen? I agree entirely with Mr Maskell who points to tiredness of the arm and a disturbance of the 'habit' pattern of stroke production as two of the reasons. Weather, too, has a marked effect in England. Cold windy days in Spring and Autumn produce a regular bi-annual crop of tennis elbow. Changes causing a greater load to fall on the elbow muscles,

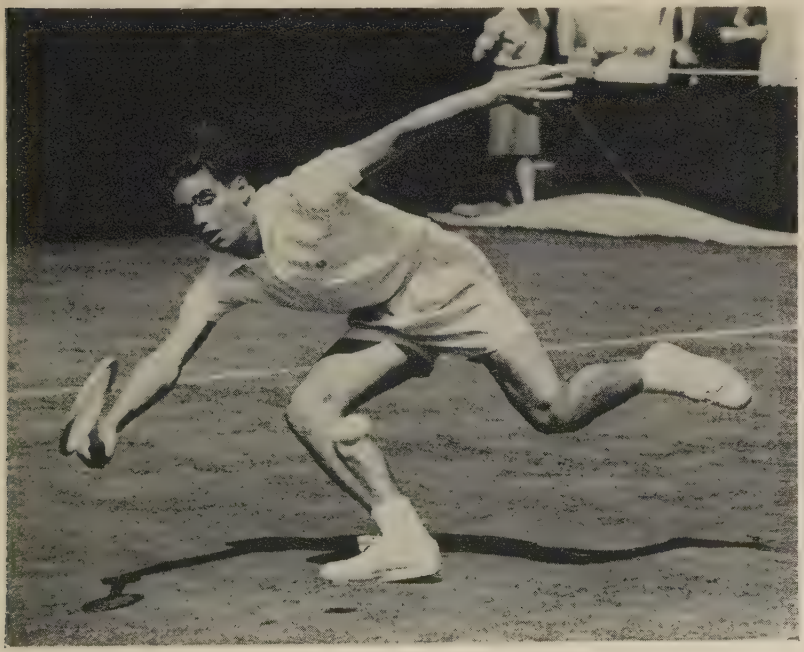


FIG. 1. Perfect body balance is necessary for both attacking and defensive shots. Unless co-ordination is exact muscle action may produce soft tissue injury.

such as the alteration in the tension of the gut, in the weight of the ball or racket, are common factors. Mistiming the ball at the beginning of the season, particularly in the production of a speedy stroke, is a frequent cause of injury.

The mechanism of tennis elbow varies with the class of player. The average club player often tells the story of an enormous mis-hit on a cold day early in the season and early in the match. The international player tells a story of a gradual tiredness of the arm over a matter of weeks and acute twinges now and then when attempting a particularly powerful stroke or on a bad bounce. This mild pain may then become gradually or suddenly worse.

Analysis of these and other factors leads to the following conclusions. Tearing of muscle fibres occurs when they are subjected to an overwhelming strain. There are three main reasons why a strain may become overwhelming.

(a) As a result of its sheer magnitude—uncommon in tennis.

(b) Because of a temporary breakdown of the normal shielding and guarding effects of the other muscles of the arm and shoulder. This factor is due to a bad bounce or mis-hit which causes an unexpected movement.

(c) A decrease in the general resistance of the muscles to strain because of fatigue or cold.

A smooth, flowing *expected* stroke is the integrated result of just the right amount of 'take' expected by one set of muscles, accurately counterbalanced by the 'give' of the opposing set. The strain or load of the stroke is cushioned by an adjustment of the tension of the muscles on which it falls.

Fatigue and cold are, in my opinion, the two most important factors causing tennis elbow. Fatigue acts by decreasing the guarding effect of the shielding muscles as well as by lessening the resistance of the actual muscles on which the strain falls. Cold acts mainly by reducing the resistance of the muscle to strain, thus rendering it far more liable to injury.

Tennis elbow is an occupational hazard. Most tennis players who play long and hard enough are liable to the injury. Illustrious names such as Hoad, Patty, Drobny and Knight show that skill is no safeguard.

TREATMENT

Tennis elbow is essentially a self-curing malady, but a severe one may take two years or longer. Moreover, many months of tedious waiting give no guarantee of certain cure, and few tennis players have the patience to wait two years. Treatment is thus necessary to speed up the process.

The first step is accurate diagnosis. Tournaments abound with professional

advice-givers who 'had a friend with it and . . .' They are an absolute menace and do a deal of well-intentioned harm. Do let a doctor see it and make a diagnosis. He can then treat it himself or elect to refer you to some specialized unit which has the experience and the facilities to carry out the right treatment. In an article of this sort I can only attempt to outline what I believe to be the right treatment. Each 'elbow' must be treated on its own merits, the problem posed by a Davis Cup player with a mild attack and a club player with the same injury being vastly different.

The sort of treatment indicated depends greatly on the severity and duration. The basis of the cure is the speeding up of the natural healing process and the limiting of its sometimes undesirable side effects such as exuberance of fibrous tissue formation which restricts free movement of the elbow.

In a mild tennis elbow speed of healing can best be insured by a regime of rest and exercise. By resting the arm I mean resting it from actual pain-producing movements. Exercise such as running, which entails the use of other limbs, is beneficial as it increases the blood flow to the injured muscle and therefore hastens the healing process. Local methods of increasing blood supply such as massage, heat and wax baths are also of value.

In the more severe case, or where speed of cure is vital, injections of hydrocortisone mixtures may work wonders. About 30 per cent of patients obtain permanent relief from a well performed injection. Injection of local anaesthetic may also be used, either with hydrocortisone or on its own. In severe chronic cases, manipulation of the elbow under a general anaesthetic sometimes produces a dramatic cure. But all these treatments necessitate expert selection and performance.

PREVENTION

Can tennis elbow be prevented? In many cases, yes.

Fatigue is a difficult factor to prevent; but sensible appreciation of the first sign of arm fatigue and the consequent easing down of tennis engagements even to the extent of cancelling one tournament can save weeks of nagging pain when it really does matter.

Many tennis elbows would never have occurred if proper warming up had been carried out. When Bannister or Brasher ran their races they often spent an hour warming up for an event which took about four minutes. Yet even tournament players walk on to a court on a cold March morning wearing scanty shorts, hit a few balls in desultory fashion for three minutes and then start to play all out. To ask cold muscles to accelerate their output from zero to maximum in three minutes is courting disaster.

Proper warming-up lasting at least twenty minutes must be carried out before stepping on to the court for match play, and a track suit is an essential aid to do it. The temporary sacrifice of elegance is appreciated but discounted.

Judicious rests in a busy season, and adequate warming up in sensible clothing, are simple measures, yet properly applied can do an enormous amount to prevent those painful tedious 'elbows'.

TENNIS ELBOW

DAN MASKELL
O.B.E.
Lawn Tennis Association Training Manager

THE injury that most commonly afflicts lawn tennis players is tennis elbow. I say this without hesitation after thirty-five years experience as player and teacher, and in close association with players of every standard from beginners to those of world class.

Tennis elbow seems to be no respecter of persons. It claims as its victims young and old, men and women, good players and bad; neither does it appear to matter whether a person plays regularly or not.

Just recently I have met two rather different, but typical, cases. The first is a boy of seventeen who has been playing since he was ten and hopes to play for his country one day. His improvement was quite marked until he was sixteen but then it became less noticeable. He is a fluent, powerful stroke-player, but has too little regard for ball-control. It was suggested to him—wrongly, I'm quite sure—that his progress had come to a standstill because of unsound stroke technique and that he needed to change his style. This he has attempted to do and the mechanics of his backhand and service are now quite different: but he has acquired a very bad tennis elbow. Pain on the outside of the elbow and weakness in gripping the racket prevent him from playing any kind of backhand shot and he also feels some discomfort when he serves. He told me that about a week or ten days after changing his style, he noticed that every time he played his whole arm became tired after thirty or forty minutes of practice, something that had never happened before. There was no pain associated with the tiredness. He first became aware of pain when he badly mishit a fast ball directed to his backhand, and from that particular shot onwards the pain has become progressively worse and his grip weaker.

The second case is of a man nearly seventy years of age who has played

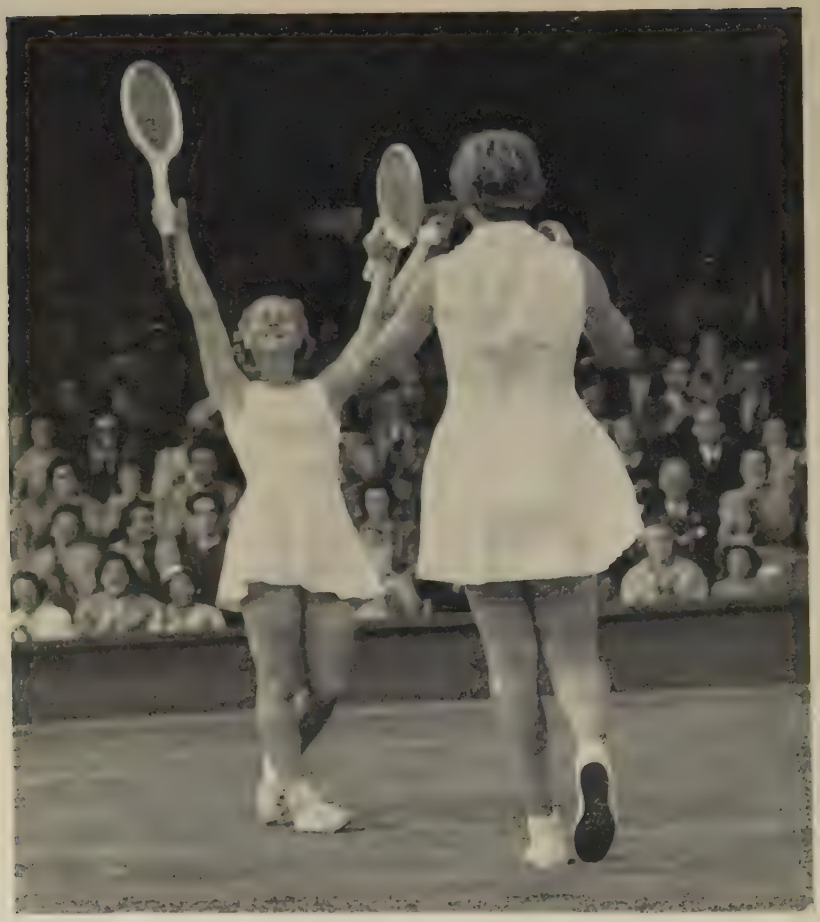
two or three times a week for fifty years 'to keep fit'. He has never been better than an ordinary club player. A few months ago he bought a new racket and for some unknown reason decided to try one with a slightly larger handle than those he had used previously. It also had tighter stringing because he felt it would 'make up for a bit of speed of shot I seemed to be losing'. This enthusiast now has tennis elbow with severe pain on the inside of the elbow whenever he plays a forehand shot. He cannot grip with any strength and finds it difficult to lift a tea-pot, or even a cup of tea. The onset of the injury has followed a fairly typical course. Our friend found that he did not altogether like the larger handle but instead of reverting to one of familiar size he compensated to a certain degree 'by gripping the handle more underneath than I used to'. He also discovered that the tighter stringing was less sympathetic to his arm and hand when he failed to hit the ball right in the middle of the racket and, possibly because he was holding his racket differently, he mistimed many more shots than he had ever done before. After a while he became conscious that a badly mistimed shot caused a certain amount of jarring and eventually he, too, was aware of weakness and tiredness in the arm after only short bouts of play. He remembers quite vividly his first actual experience of pain 'when he tried to whip up a low awkward ball on the forehand'. He did not play at all for two weeks thinking the rest would do his arm good: but immediately on resuming play he noticed the pain was as bad and seemed to have come to stay.

Both these cases, though different in detail, have, I think three common factors:—

1. a fairly sudden change of 'habit' performance,
2. the gradual onset of weakness or tiredness in the arm, and
3. the injury occurred on a particular shot and from then on play was impossible without pain.

My own tennis elbow, which occurred some nine or ten years ago and kept me out of serious tennis for four-and-a-half months is, once again, different in detail, but fundamentally the same. The actual injury did not, however, take place on a tennis court.

Fred Perry (who won the Wimbledon Championship in 1934, 1935 and 1936), and I were on a four-week tour of the country. We gave a two-hour coaching demonstration session afternoon and evening which consisted of demonstrating basic strokes and their variations, elementary and advanced tactics, etc., finally finishing with one or two sets of hard singles play. It was my task to demonstrate the different grips, swings and spins. Our itinerary was extensive and we drove our own cars carrying the necessary equipment.



Many of the small towns we visited lacked a tennis court with a true playing surface and by the beginning of the fourth and last week my playing arm felt heavy and tired.

The actual injury occurred a couple of days later. I was driving and almost passed the concealed entrance to the club at which we were due. I hastily jerked the steering wheel to the right; and I felt as though I had torn something in my elbow. The pain was acute for the moment but I was able to play that day, though in a very guarded fashion, especially on the backhand. The outside of the elbow became more painful and sensitive to even the lightest touch. I could not grip for a backhand shot and contact with the ball when

serving made me wince. The last performances on the tour were full of compromise. I was only able to 'feed' the ball up to Fred Perry with my forehand while he nobly did the rest!

The sequence I have described above is again evident: disturbance of the 'habit' pattern, in order to demonstrate variations of ordinary, everyday basic strokes: then tiredness (caused by a combination of excessive play on untrue, indifferent surfaces, together with far too much car driving): and, finally, the actual injury when performing in an emergency an unusual and sudden arm action.

The treatment given me at first was quite unsuccessful. I had my elbow bandaged and was advised to play, ignoring the pain, until the injury healed itself. I was also given a form of heat treatment about three times a week. This 'cure' had to be abandoned after a week or so, simply because I could not endure the pain when attempting a backhand, and to a lesser extent, the service—and the whole arm seemed to be getting weaker every day.

The treatment next prescribed was possible to carry out and eventually proved successful. I was advised to play a little each day, without any form of bandage, but to avoid any stroke that caused pain. This meant that I was limited to shots on the forehand side only and, at first, I restricted myself to an hour before lunch and about the same afterwards. The heat treatment was stopped.

As the days went by I felt no ill-effects and began to play about three hours a day. The outside of the elbow became less sensitive to the touch and the arm felt generally stronger until, eventually, I was able to stroke my backhand and service gently without discomfort. In all, it took four and a half months before I could play serious tennis, and even then I was conscious, at times, of slightly less strength in my backhand grip.

There was a time when Perry also might have contracted this tennis 'bogey'. In his late teens, when he was full of high promise and ambition, he set himself the admirable task of taking the ball 'on the rise'. He already took the ball earlier than most; but now he attempted to take it even nearer its pitch. Our practice sessions normally lasted two hours and in that time he would hit about 3,000 shots. Really 'early ball' play demands perfect timing; and during this part of Perry's apprenticeship he mishit a great many shots, with the result that the constant jarring was beginning to cause a weary arm. Had he gone on trying to 'gild the lily' I am sure he would have been risking tennis elbow. As it was, being a practical person, he compromised by taking the ball a little later, thus giving himself some margin for error, but still taking the ball consistently earlier than almost anybody else playing at the time, or, perhaps, since.

15

MOTOR RACING ACCIDENTS

Dr W. CARTON WINTERBOTTOM

Deputy Senior Medical Officer, British Racing Drivers' Club
Senior Medical Officer to British Racing and Sports Car Club, Silverstone Circuit

THE SERIOUS accidents of racing usually cause fractures of the limbs which are no different from those encountered in any other sport. The special danger is the head injury and this becomes more common and more dangerous as lap speeds rise.

CONCUSSION

If there are no limb bones broken, despite damage to the car it is nearly always because the driver is thrown clear of the vehicle. He is very apt to stand up at once and pronounce himself all right, only to show, in a very short space of time, that he is suffering from concussion. Time and again drivers do this and then within another sixty to ninety seconds they are prone on the ground. Such patients obviously need observation and they should always be taken to the Medical Headquarters.

When the head injury is more serious the injured driver makes no attempt to move and has to be lifted from the driving seat or, if he has been thrown out he remains inert where he landed. This type of casualty should only be moved by ambulance.

There are two main causes of accidents—mechanical failure or error of judgement. As it is impossible to eliminate these there will always be accidents, although everything is done to make motor racing as safe as possible in this country. At Silverstone the spectators are protected by a ditch and a mud wall.

As most people know cars are carefully inspected ('scrutineered' in the language of racing) before practice starts at any meeting. Drivers are not allowed to practice unless a medical officer is present. What may not be widely known is that drivers must produce to the scrutineers a medical certificate of fitness. Without this they cannot drive. Crash helmets are also compulsory.

A word about the average medical organisation on the motor racing circuits

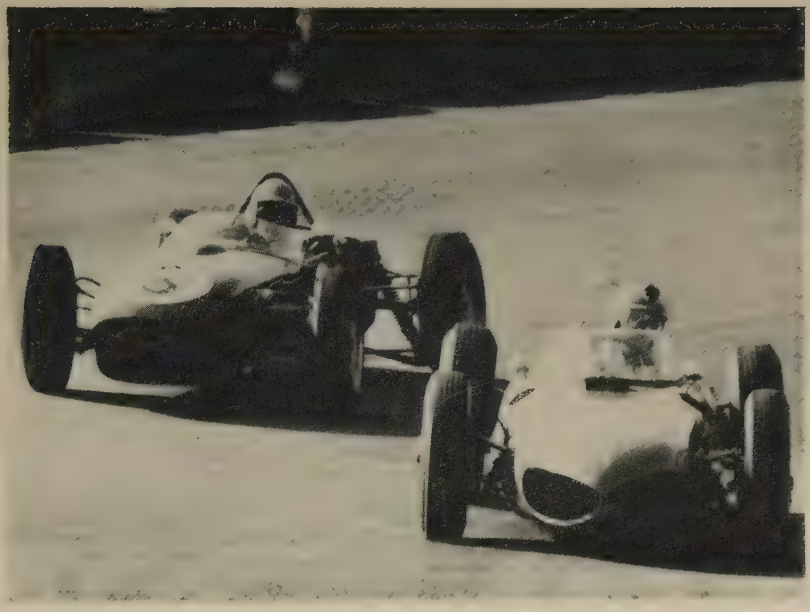


FIG. 1. With both these cars travelling at very high speed a minute error of judgement could be responsible for a major accident.

in this country. There is usually some sort of first aid post. Silverstone has a very adequate first aid headquarters which is a permanent building. This is equipped for first-aid attention and minor surgery, but is only to be regarded as a clearing station. Any injured driver is always taken to a general hospital.

The medical headquarters is in the charge of a chief medical officer who is in telephonic communication with Race Control. Doctors and ambulances are placed at vantage points round the circuit, and all are well briefed on what they should do if there is an accident.

All these arrangements help to promote the safety of both drivers and spectators; but many doctors with experience of motor racing feel that a driver involved in a severe accident should be forbidden to drive again for a minimum of a week, whether or not he showed any signs of injury at the time. There is the possibility of delayed shock, which, at high speed might incapacitate a driver sufficiently to cause another serious accident.

16

INJURIES IN ROWING

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Rowing Team Manager for Great Britain

INJURIES attributable to rowing are not numerous and are for the most part the results of faults of technique. They are not accidents in the ordinary sense of the word, and are in most cases preventable. Nevertheless they do occur quite frequently; and once they have happened the length of time required to recover and the probability of recurrence is too great to allow the individual to continue rowing in the crew in many cases.

Heart conditions. There has been for many years, a popular superstition that rowing is sometimes the cause of 'strained' or damaged hearts. It can be stated quite definitely that nobody has ever caused any damage to his heart by rowing, provided that there was nothing wrong with it when he started and that he did not continue to row at any time when he was physically unfit. By this I mean that all oarsmen should be medically examined before starting training (to ensure that they have no cardiac or any other abnormality) and that under no circumstances should they row while suffering from or recovering from any illness, especially influenza or any other pyrexial condition. If these simple precautions are observed there is no danger of heart damage.

Muscular strains. These are by no means uncommon and the muscles affected are those of the shoulder, back and abdomen.

Damage to the shoulder and back muscles is invariably caused by faulty technique and results from 'over-reaching' at the end of the forward swing, thereby taking the weight at the beginning with the shoulders or back before the power from the legs comes into operation. The outside shoulder is especially liable to strain if it follows the oar handle round on the forward swing rather than swinging straight down the boat. The shoulders should be kept down and loose, and the forward movement maintained as a swing from the hips, not as a reach forward, so that the beginning is taken directly with the spring from the legs transmitted 'through' the shoulders and back, rather than initiated 'by' them.

Strained abdominal muscles also occur at times, usually in the early stages

of training, being the result of either too much rowing before the muscles have had a chance to become strong enough, or of lying too far back at the finish. Usually both these factors are combined, for the effect of difficulty in getting the blade through is either to lie back at the finish or tear the blade out.

An attempt to cover too long distances in the early stages, before the muscles are fit enough to manage it, may be the fundamental cause of faults which produce muscular damage. Exercises out of the boat, in the form of properly graduated circuit training, especially at the beginning of training, can do much to prevent these troubles, provided the exercises are carefully thought out and not allowed to become an end in themselves.

Tenosynovitis. This is a very common rowing injury, especially in the early stages of training, and is in all cases the result of gripping the oar handle, usually with the inside hand.

Once tenosynovitis has started, rowing is bound to aggravate it. Even slight tenosynovitis makes the hand grip tighter on the oar, worsens the condition, and creates a vicious circle. The time required for cure can usually be measured in weeks rather than in days, and unless the cure is complete before rowing starts again, a recurrence is certain. Indeed, even after an apparently complete cure the condition may relapse during the ensuing weeks. The only satisfactory cure is prevention. The oar must be held lightly in the fingers and not gripped at any part of the stroke, especially the finish, where the turning movement which causes the trouble occurs.

Blisters. These are mainly on the hands and usually on the fingers. If they occur on the palm, the technique is faulty: the oar is being gripped in the hand rather than held lightly in the fingers. Blisters are commonest in the early stages of training, before the hands have toughened. The most important part of the treatment is preventive, the correction of the oar holding technique. The skin of the hands may be hardened by the application of surgical spirit (though this is not always successful and should not be persisted in if it does not seem to be helping). The blisters themselves should be carefully treated with perfect sterile technique, especially if the skin is broken. Mercurochrome or Flavine are suitable antiseptics to apply to broken skin. Blisters should only be opened under proper aseptic surgical conditions, and never by a prick with a non-sterile needle. A simple blister almost invariably responds to simple treatment, but an infected blister may cost a man his place in the crew. To cover blisters on the hand with bandages, or any other form of covering, except for a very small area, is useless, as the covering only moves on the blister and aggravates it. Elastoplast, even directly onto a skin, is usually only effective for a brief time since it soon rucks up into folds and causes further trouble.

In wet weather the skin of the hands may become sodden, softened and more liable to blister, even during the later stages of training. Blisters may also occur on the heels, mainly in very wet and cold weather, where clogs are being used, due to the wet sock rubbing against the heel trap. Elastoplast may be applied directly over the damaged skin, for the heel does not move enough to displace it. Padding, if necessary, can be applied over it.

Lumbar disc lesion. This is not a common rowing injury, though many cases of strained or torn back muscles have been diagnosed as a 'slipped' disc. The back muscles of oarsmen are usually very strong, and therefore injury of an intervertebral disc is rare; but a disc occasionally is damaged when the boat is lifted out of the water, especially if the water is low. If the crews are taught to keep the back firm as they lift the boat, in the same way as they take the beginning of a stroke, the accident should not happen.

Abrasions to the back of the calf. One often sees these, which occur when the knees are held down flat and the back of the legs are kept pressed against the front ends of the slide runners. This is not due to any fault in technique, but is due to a fault in the rig of the boat. It can, in most cases, be cured by alterations to the length of the stretcher and the height of the seat.

FIG. 1. Co-ordination and training should prevent any undue muscle strain.

Sore bottom. This is much less common since fixed seats were abandoned, but it does still occur and, although not usually serious in itself, it can be painful enough to make the oarsman sit in a crooked position in the boat and thereby strain a muscle. The cause is usually continued rowing on a wet seat or in wet shorts, or the wearing of shorts made of non-absorbable material. There should always be plenty of dry shorts available, made of flannel or wool, if this trouble is to be avoided.

Lastly it must be remembered that all the members of the crew differ in some respects in their physique, whether in height, length of leg, body or arms; and quite small alterations of the rigs to take account of these individual differences may often prevent development of a faulty technique, which may in turn be the cause of a physical injury. Rowing, more than any other sport consists of a series of repetitive movements from which relief can only be obtained by the introduction of some alteration, or fault, into the technique; and therefore, once an injury has occurred it will be perpetuated and made worse by every stroke. For this reason also, an injury, once sustained, is very liable to occur again, even after considerable absence from rowing and apparent cure, unless the cause has been found and eradicated.

A rowing injury is not an accident, it is a result of a faulty technique, inappropriate individual rig, or lack of common sense; and a proper appreciation of this, both by the oarsman and the coach, is the best means of prevention.

17

RUGBY FOOTBALL

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Ireland, Barbarians, British Lions

RUGBY football is usually a hard and friendly tussle between the two teams. The advantages of playing the game far outweigh the risk of injury. Fortunately this risk is an extremely small one when compared with the number who indulge in the game. Nevertheless, it is worth considering and analysing these injuries so that all possible preventive measures may be taken to decrease them.

There have been very few reviews of rugby football injuries. The best one I know is by O'Connell of St. Vincent's Hospital, Dublin, writing in the *Irish Medical Association Journal* of January, 1954.

In it he reviews 600 cases. I am grateful to him for allowing me to quote from the paper. It confirms many of my own impressions.

This is his general analysis of all injuries:—

	Cases	*Percentage of all injuries*
Head injuries	129	21·5
Neck injuries	14	2·33
Upper limb (shoulder)	110	18·33
Upper limb (remainder)	77	12·83
Thorax	17	2·83
Abdomen	15	2·5
Spine and pelvis	35	5·83
Lower limb (fractures all bones)	53	8·83
Femoral muscle injuries	17	4·5
Knee (ligaments and cartilage)	75	12·5
Calf muscles	6	1·0
Ankle ligaments	42	7·0
Total	590	99·98

It is interesting to note roughly the different types of injury which are associated with different positions:—

Forwards receive most of the injuries around the head and face, especially the ears.

The scrum-half suffers most from injuries to the hands and face.

The three-quarters are most likely to receive shoulder and leg injuries.

The full-back is the least liable to injury on the field.

Of this total of injuries occurring over twenty years, five were fatal. Three were fracture dislocations of the cervical spine, one was rupture of the spleen and one was from tetanus. Of all the other injuries the great majority made a complete recovery and resumed playing the game.

This accords with my own experience of about fifteen years contact with rugby football. The most serious injury is the fracture—dislocation of the cervical spine. I have known of two fatal cases and one left with some loss of activity and sensation in the limbs.

Minor dislocations or subluxations occurred in three others, but these all made a good recovery. No other fatal injury or injury leaving permanent damage has occurred in my experience and I have never come across a severe intra-abdominal injury.

Is there anything that may be done to prevent this rare, though most serious of all rugby injuries? All O'Connell's fatal cases were due to charging or collapse of the scrum—the injury is caused by the neck being acutely flexed or bent towards the chest. I used to see many more scrums collapse than I do now. This is due, I believe, to:—

(a) the fairly recent laws concerning front row binding

(b) the front row coming together somewhat more gently, and

(c) the general vigilance of referees in watching for collapse of a scrum and stopping play immediately.

In the cervical fracture dislocations of my experience the three serious ones were also all scrummage injuries. In these injuries I know that at least two of the players were in very poor training and one of those who died was brought on to play at the last moment, although he had not been in rugby togs for at least a month.

This is surely worth our attention and a general rule could be laid down for us all. *No player should join in a game unless he has done a reasonable amount of training.*

Forwards, especially, should practise neck exercises, for example pulling away from another player whose arms are clasped round his neck, and scrum-

maging against a single individual. These should help to improve the muscle tone, which is so important in preventing any dislocation.

It is impossible to discuss all the injuries in detail but it is worth noting the very high number of injuries to the shoulder region. These make up 18 per cent of all injuries received, and in the experience of O'Connell and myself are among the most incapacitating. Between us, we know of at least half a dozen international players who have been laid off the game for long periods, one permanently. These injuries take a long time to recover completely, and some of the acromio-clavicular subluxations and dislocations which I have seen never returned completely to normal and the player was left vulnerable to further injury in that region.

It is not in the nature of rugby footballers to wear protective apparatus of any description. Nevertheless, this region of the body is the one most easily and more effectively protected than any other. In view of the fact that one in five of all injuries occurring in rugby players are in this area I believe that a minor change of attitude towards a simple device for protecting the shoulder is required. O'Connell recommends a piece of sorbo-rubber ½ in. thick, 8 in. long and 6 in. wide slipped into a pocket in the jersey lying over the shoulder. It is not suggested that large thick leather pads should be worn, because these give the wearer an unfair advantage over an unprotected opponent and would only make for rougher play and harder tackling.

This sort of protection is almost unnoticeable and would save many players from this most annoying of all rugby injuries.

I have a few final suggestions to make:—

1. The time has surely arrived in this country for the proper padding of all rugby posts and the provision of pliable touch flags made of yielding rubber or some such substance. O'Connell has noted twenty injuries from collision with these objects, some of them serious —including fracture of the skull and concussion. We could take a lead from New Zealand rugby in this direction.

2. One person on the field of play should look at all injuries—preferably someone with a medical background. At the same time a doctor on the touch-line should be ready to come to the spot at the wave of a hand from the person seeing them. It is hardly fair to place the responsibility of deciding whether a player should continue playing with an injury on a captain or other individual who has no medical knowledge and may not know how serious the injury may be.

Naturally at many games a doctor may not be available. Here the rule should be —if there is any doubt about the injury then the player involved must not be allowed to continue.

I know of a player who continued to play with a subluxation of the cervical spine. One shudders to think what would have happened if he had received a further severe knock. The death from ruptured spleen mentioned by O'Connell was undiagnosed—probably not seen by a doctor.

A few words about the player who insists on playing on despite injury. If it were possible to produce statistics showing how many sides with fourteen men have won the game in which they had one man on the touch-line, I am reasonably sure that the balance would be in favour of the fourteen men.

This is likely to be due to the fact that the fourteen men play the 'shirt off their backs'. On the other hand it is my own impression that very many games are lost by fourteen fit men and one injured man, who has returned after receiving attention but is incapable of giving any real assistance to his side. We see this player so frequently being sent out to the wing position. Most of us like to think that, even when injured, we are of some use to our side. This idea must be abandoned. Not only are we of no assistance but we are a liability to the other players, who know they cannot depend on us and try to do our job as well as their own. It is our duty not to return to the field, if we are not going to help the side. If, having returned, we realise how handicapped we are, then we must leave the field.

The captain of the side should also appreciate that one unfit man in the side is a tremendous handicap and that he is more likely to lose the game with the player on the field than off it. He should then advise the player injured to leave the field for his own good and the good of his side.

3. Finally, injuries are prevented by training, not forgetting the gentle warm-up before a game.

RUGBY FOOTBALL

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DIRECT INJURIES in rugby are often the result of a collision of two or more players (not always on opposing sides). The man making a 'mark' is always likely to be tackled hard while his body is fully extended to catch the ball. He may be badly 'winded', but rarely receives a serious injury. This shows that it is not always the violence of an impact, but the way in which it is received that determines whether it will result in injury.

In the line out, as elbows flail in an attempt to stop the opposing forwards

FIG. 1. Obviously in Rugby Football there must be some risk of accidental injury.

getting past, nose-bleeds are common; and eyebrows are often cut both in the line out and the scrum, where heads are liable to collide. 'Cauliflower ears' usually start as the result of a direct blow, followed by continuous rubbing against the bodies of the other forwards as they bind together in the scrum.

A spectator unfamiliar with the game would imagine that fractures were common, but in fact they are rare. They occur mainly when a player in a vulnerable and awkward position—perhaps having fallen over someone on the ground—is himself fallen upon. The most common break is in the lower leg, a lesion often received by a man dribbling, when an opposing player, in falling on the ball, also falls across his leg and traps his foot. The dribbler, unable to arrest his momentum, continues forward, snapping his leg. I have witnessed this personally several times.

Dislocated bones, particularly of the fingers, are fairly common, when one person tries to handle the ball as another decides to kick it: and bruises and abrasions are bound to happen in a game in which hard knocks and hard grounds are so often associated.

Indirect injuries such as pulled muscles occur mainly at the beginning of a game, and more so at the beginning of a season when players are not trained and ready for the effort required of them.

Shoulder and neck muscle injuries affect usually the forwards, as a result of the pressure and strains of the scrum; but a tackler who has the misfortune to put his head on the wrong side of a runner's legs may be similarly damaged, directly by a blow or indirectly in the subsequent fall.

Knee and quadriceps injuries are mainly the result of too sharp a turn especially when the ground is waterlogged so that the boots are inclined to stick while the legs move. Injuries to these muscles as a rule take longer to heal than any other, for the legs have all the weight of the body to support.

The injury that causes the team secretary the most worry, however, is the simple muscle bruise. Sometimes it passes very quickly; but Sunday morning often finds a player almost unable to get out of bed because of a late muscle reaction.

Prevention. Injuries of any sort are most likely when a player is cold or tired, his reactions being then at their slowest; and this is usually at the start or finish of a game. They can best be prevented by a gradual warm-up before the team leaves the changing room. Players should also learn to rest and relax when the play allows them a short respite.

Training of course plays a big part in any game: and the better a player has prepared his mind and body the less likely he is to be injured.

18

SWIMMING

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British Swimming Team, European Games 1947
British Swimming Team, Olympic Games 1948
British Empire Games 1950 (Gold Medal)
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APART from the hazards of getting into and out of the water, swimming is one of the few sports in which it is difficult to injure the body. Exceptional contortions may be performed without the slightest strain, for the body is supported by the water so the effects of gravity are therefore totally eliminated. Furthermore, with the exception of water-polo, there should be no physical contact between the competing athletes.

Another factor of considerable importance which contributes towards the relative rarity of injuries amongst competition swimmers, is the preponderance of extremely youthful participants in the sport: indeed, it is unusual to see a champion in whom the epiphyses are completely fused. The championship swimmer is an obsessional, highly-strung adolescent, who must cultivate an extremely high degree of fitness if he is to sustain any measure of success. Modern training techniques insist upon the out-of-water development of many individual muscle groups, as well as many hours and miles of water training, to be undertaken daily. He is highly sensitive and at the merest suggestion of illness or injury will seek aid immediately. This usually aids both diagnosis and treatment, the subsequent period of disability being appreciably reduced as a result. Serious injuries are, therefore, very few and far between. Fractures occurring as a direct result of the sport are seldom reported. Sprained joints are unusual, but minor muscle and tendon strains are relatively common. These occur predominantly around the shoulder joint.

'*Swimmers shoulder*'

Probably the most important factor in the production of this lesion is the form of arm recovery necessitated by the front crawl. At the end of the propulsive stroke, the arm ends up at the side of the trunk with the hand by the

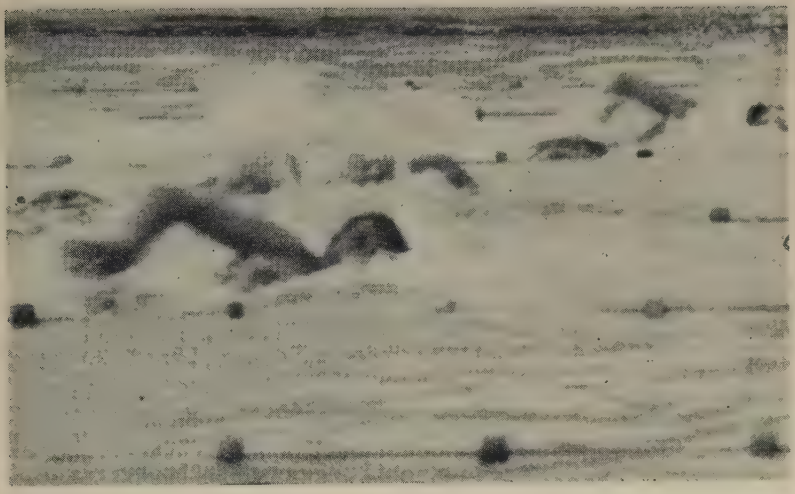

FIG. 1. Photograph showing the awkward position of shoulder during the movement of recovery.

FIG. 2. Diagram illustrating the position of the shoulder muscles at the initial phase of recovery. (A) Supraspinatus; (B) Interscapular muscles.

thigh, the shoulder being fully adducted and in a position of mid-rotation. The hand is then lifted from the water, the scapula forcefully retracted, the shoulder rapidly externally rotated and then abducted. As the arm comes to a position level with the head the shoulder is partially internally rotated: which

brings the arm with the elbow extended ahead of the swimmer, in line with the shoulder and in the correct position to commence the next downstroke. This movement is helped to some extent by body roll. The majority of variations of the front crawl stroke have derived as a result of attempts to accomplish the recovery of the arm with the minimum of effort and maximum of comfort and relaxation, and the elimination of 'roll'.

Trunk roll was taken to the extreme by the Hungarians in the European Games of 1947 and the Olympic Games of 1948; recovery was accompanied by an exaggerated lunge of the opposite shoulder into the water, the recovering arm being simultaneously 'flung' over. Despite such variations, recovery remains an extremely awkward movement and places considerable strain on the scapular muscles, abductors and rotators of the shoulder joint, for the first few degrees of movement when they are acting in a position of maximum effort yet minimum efficiency (see Figs. 1 and 2).

The majority of swimmers suffering from painful shoulders have one or a combination of two main lesions.

1. Minor sprains of the muscles of the gleno-humeral and interscapular groups.

2. Supraspinatus tendonitis.

Ruptures of a few fibres of these muscles are occasionally seen, but this usually occurs in the older age groups and is associated with some definite strain, for instance a missed turn during a sprint.

Breaststrokers tend to suffer from strain of the trapezius alone, particularly when fatigued. Quite severe strains can also follow diving into the water with the head forced forward.

After treatment for these shoulder lesions it is vital that the technique be checked closely, training recommenced slowly and built up gradually.

Leg injuries

In front and back crawl the leg action is a powerful up-and-down scissors kick produced essentially by repeated flexion and extension of the hip joint; flexion producing the forward thrust. Flexion of the hip is accompanied by a powerful extension of the knee joint from a partially flexed position and a forced dorsiflexion and inversion of the foot, the whole movement resulting in a 'whiplash' action. The main muscle groups used to produce this strong thrust are the quadriceps and hip flexors, particularly the psoas, iliacus and rectus femoris. As the movement is repetitive and essentially rhythmic without any 'jerkiness', muscles tears or ruptures are seldom encountered.

Occasionally minor strains of the quadriceps are seen in swimmers who

FIG. 3. Swimming is essentially a rhythmic balanced action and accidental injury is unusual in top class swimmers: this applies particularly to back stroke

indulge in excessive periods of leg work when training, but even this is usually associated with a certain degree of unfitness.

The breast stroke swimmer utilises a totally different form of leg stroke. The thrust is obtained by a vigorous adduction of both legs with full extension of the hips, knees and ankles from a position of wide abduction. As a result of this exceptionally powerful and relatively unnatural movement, two main injuries are seen. The first of these is a strain or even occasionally a tear of fibres of the hip adductor muscles, usually the adductor longus. This results in a clinical picture similar to 'horse rider's strain', but usually not so severe. The muscle is seldom ruptured but may be tender, thickened and accompanied by pain on resisted hip abduction. It is usually unilateral. As a great percentage of the driving power of breaststroke is derived from the leg stroke, this strain renders hard swimming impossible.

The other leg injuries occasionally seen in breaststrokers are a minor tear of the medial ligament of the knee joint and a strain of the coronary ligaments of the medial meniscus. As the legs are forcibly adducted, a forced adduction of the knee joint occurs: this results in a forced separation of the medial articular surface of the tibia from the medial femoral condyle. Such injury is

again more commonly seen in the unfit swimmer with reduced muscle tone, who is putting in a violent effort during the final stages of a race.

Turning injuries

The essential features of a correct racing turn are to reverse the direction of swimming and obtain the maximum possible distance on the 'push off' in the minimum possible time. Whilst racing or during timed training, hurried or careless contact between the hands and the tiled, metal or cement walls of the pool may cause minor injuries. Traumatic synovitis of the interphalangeal joints may follow contact with the wall when the fingers are extended. Fractured shafts of the phalanges are more unusual, but sprains of the wrist joint and wrist flexors occur commonly, particularly in breaststrokers. Prevention of these injuries can only be accomplished by ensuring that the swimmer is familiar with the pool in which he is to race. As most 'turning ends' vary in design, careful inspection from the side of the pool before entering is well advised.

DIVING INJURIES

Injury to divers is, somewhat surprisingly, comparatively unusual. It is not until the diver attains heights of ten metres or more that he enters the water with any great force. From boards of this height, the speed of approximately twenty five miles per hour is approached and a depth of up to sixteen feet reached, after entry. Fortunately the novice will seldom venture to this height even to jump off, and the experienced diver will usually learn how to enter the water correctly before doing so.

In order to enter the water comfortably, the diver 'stretches out' a few feet above it, entering either feet first or with the fingers, wrists and elbows extended and together, the shoulders fully abducted and the head tucked between the arms. Failure to adopt this position may result in strains of numerous muscle groups. The flexors of the forearm, the triceps and the trapezius are particularly prone to hyperextension injuries.

Fractures of the fingers, radial styloid and carpal bones have followed striking the bottom of the pool hard, and abrasions of the back from grazing the diving board or even the side of the pool during an incorrect descent.

Several cases of an unusual diving injury have been reported by Bleasdale (1954). These are 'blast' injuries to the chest wall and lungs, which follow hitting the water absolutely flat from the high boards. The sudden pressure changes are sufficient to result in severe shock and minor haemoptysis: epistaxis and bilateral orbital haematomata may also occur.

Injuries sustained by water polo players include all those described for swimmers, together with those that result from contact between the players. Fractures of the nasal bones and extensive contusions of the face and orbit occur when the player is struck in the face by the ball. Lacerations of the face are commonplace and, on more than one occasion, an abrasion of the cornea has resulted in the loss of an eye. Quite severe abrasions of the trunk and upper limbs are frequently seen and the author feels that there is a definite case for routine examination of the players' nails by the referee.

GENERAL

All competitors in water sports are prone to sinusitis; back stroke swimmers and divers seem especially liable. Chlorinated water is inhaled or forced into the paranasal sinuses, particularly the maxillary antra; and a low grade irritant inflammatory sinusitis results. The mucosa becomes very sensitive and secondary infection follows easily. For this reason the majority of backstrokers now wear nose clips. Unfortunately this precaution is impractical for divers and front crawl swimmers.

Tinea pedis ('Athlete's foot') is almost endemic amongst swimmers. Thirty per cent of the 1952 British Olympic team suffered from this condition, which frequently results in appreciable disability if it is permitted to go untreated. Despite absolute cleanliness, the use of foot powder and frequent changes of socks, many swimmers are subject to chronic fungus infections, particularly of the toenails and at present it seems quite impossible to prevent repeated re-infection in the swimmer who trains regularly.

REFERENCE

Bleasdale, N. (1954) *Med. World.* Lond. 80, 137.

19

WATER SKI-ING

The late AUSTIN T. MOORE

M.D.

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WATER SKI-ING, in a relatively short time, has become one of the most popular sports in the world. Its origin is claimed, about 1928, both in the North-east and North-west portions of America. Rapidly it became popularized in southern areas with warmer climates and Cypress Gardens, Florida has become known as the water ski capital of the world.

In Cypress Gardens a water ski show is put on four times daily. A troup of trained, expert, professional water skiers, demonstrate jumping, kite flying, bare foot ski-ing, multiple ski-ing in pyramids, forward, backward and sidewise ski-ing on 'banana' skis, disk riding and many other amazing tricks and stunts. Visitors from all over the world come to these water shows.

The American Water Ski Association with its headquarters in Winter Haven, Florida was born in 1939. Its express purpose is to formulate rules under which sanctioned tournaments may be conducted, to encourage non-competitive recreational water ski-ing, to make available literature and materials that will develop the sport, improve the capabilities of participants, and prevent accidents or injuries.

The first National Water Ski Tournament was held at Jones Beach, New York in 1939, under the sanction of the American Water Ski Association. Except for war years, a national tournament has been held each year since 1939.

The International Water Ski Union was formed in 1949 and that year the first international contest was held at Juan Les Pins, France. The first world championship was established at that time. National champions and other competitors came from the United States, France, Belgium, Italy and other European countries. Injuries and serious accidents among professional water skiers have been remarkably low.

Water ski-ing is a sport that can be enjoyed by every member of the family. Most people everywhere are attracted to water resort areas where there is swimming, diving, beach bathing, boating, surf board riding, etc. Water ski-

FIG. 1. Water ski-ing is an enjoyable sport which is increasing in popularity.

ing has tremendously increased the sale of boats, especially the smaller outboard motor boats.

People in warmer climates, in their anxiety to get to water, will construct small ponds, build cottages on lakes or maintain homes at water resort areas. On week-ends the highways to resorts are filled with cars towing trailers with boats, skis, fishing equipment and other paraphernalia. Many of the laughing, fun seeking occupants of these cars do not realize the hazards of water ski-ing. Too many skiers in an area can lead to accidents and ski-ing in unfamiliar water can be dangerous.

The dangers of snow ski-ing are well known and an accurate estimate of accidents can be determined through snow ski patrols. It is impossible to calculate accidents and injuries resulting from water ski-ing. Those of us who live in resort areas and treat these injuries know that the accident rate is high, fatalities have occurred and water ski-ing injuries are perhaps more frequent than those that take place in snow ski-ing.

In snow ski-ing the participants are hardy athletic people. They condition themselves before beginning, they take exercise and 'warm up' before getting on skis, they practise on gentle slopes. Only the most skilful venture on the steep ski runs. Distances on land and in wooded areas can easily be estimated and, above all, there are instructors, guides and supervisors who see to it that well established rules and regulations are understood. The ski tow is the great temptation of snow skiers to go to elevations beyond their capabilities.

There are no rules to prevent an individual from putting on water skis. He climbs out of the back of a boat on a cold chilly day, he may not be wearing a life belt, he may not even be able to swim, there may be only an inexperienced driver in the boat, who sits on the back of the seat or the side of the boat and can scarcely wait to 'hit it'. Under such circumstances, a disaster is being invited. Distances are difficult to judge over water, there may be floating or sunken objects in the water, piers, boat houses or other boats may be near by and there are many attendant conditions that make accidents or injuries likely to occur.

LESSONS FOR THE BEGINNER

Education, safety programmes and legislation to enforce safety measures have not kept pace with the fantastic rapidity with which water ski-ing has developed. The novice should be sure that he is outfitted with the proper equipment and he should be sure that the boat driver is experienced and considerate. A large person requires long, wide skis to support his body weight and enable him to come up out of the water promptly. The boat motor

FIG. 2. Water Ski-ing for children is permitted at relatively low speeds.

must have sufficient power. A 25 horse power motor may operate satisfactorily for children but a fifty or seventy-five horse power motor is better suited to adults. A faster speed is required for heavier individuals. Ski-ing may be practical at 12-18 miles per hour; about 25 miles per hour is the average speed for amateurs. Thirty-five to forty-five miles per hour is required in stunting, jumping and barefoot ski-ing. At times in racing, or 'cracking the whip' speeds of sixty or more miles per hour may be attained. The higher the speed the greater the momentum, the harder the surface of the water by comparison and the greater the likelihood of injury. Originally tennis shoes were fastened to skis and ankle injuries were frequent. Now adjustable foot pieces are made of moulded rubber. These grip the feet firmly during the take off and while ski-ing but they slip off in case of an accident. The skier should be sure that the skis and foot plates fit properly. He uses a stout nylon tow rope that is 75 to 100 feet long and he should be sure that it floats freely, and is not entangled in any object or any part of his body. He should always wear a life belt and he may wear a detachable flag or marker to give warning to other boats in case he falls in the water.

The beginner should be in good physical condition, he should be warmed up and limbered up before beginning to ski and he should not attempt to ski when fatigued. He learns to ski on two skis. Later he may progress to the slalom and later, as confidence of balance is obtained, he may be able to stand on one foot on the dock, jump in the water at the proper instant as the boat takes off and come up standing on only one ski. Still later he learns to ski forward, backward or sidewise and to use the disk. It is easy to understand that all of the manoeuvres are hazardous and the beginner must be careful to avoid injury. The driver and the skier should have a complete understanding that they are to stay a safe distance from objects in the water, other skiers, other boats and docks or piers about the water's edge. The beginner should avoid sharp turns, he should come in to land slowly and be parallel to docks or the water's edge.

TYPE OF INJURY

Injuries usually result from one or more of the following causes:

1. Falling in the water
2. Striking some solid object
3. Becoming entangled in one's own or another skier's tow rope
4. Being struck or run down by a boat.

Falling in the water is the most frequent cause of sprains, joint dislocations

and tears of muscles, tendons or ligaments. Damage is also done by forceful entry of water into body cavities. Ankle injuries are not as common as in snow ski-ing but sprains or fractures can occur when a water skier falls when coming in for a landing.

When falling into the water the tendency is to put out the hands, arms or legs to protect oneself. This is the worst thing the skier can do. The skier should learn to sit down or roll like a ball. The spread eagle position frequently causes dislocation of the shoulder and other joints. The knee may be dislocated and ligaments of the knee are frequently torn or the semilunar cartilage fractured. When the skier loses control and the skis leave their normally paralleled position torsion stress can produce fractures of the leg bones or femur. The author has treated a torsion fracture of the femur due to a fall in water. Knee joint dislocations can be very severe with danger to the circulation. Amputations have been necessary. Hip joint dislocations can occur. The author had one case of severely dislocated knee joint where the circulation was questionable. Open surgery was performed to repair the torn cruciates and internal collateral ligaments. The internal semi-lunar cartilage was badly torn and was removed. The circulation re-established itself.

The worst falling accidents are fracture-dislocations of the cervical spine due to falling forward on the head after coming in for a landing at high speed. The author knows of two injuries of this nature that ended fatally. A ruptured supraspinatus attachment or a torn musculotendinous cuff are common injuries to the shoulder. The fingers may be sprained or dislocated in falls, especially at the time of landing. Hamstring muscles and plantaris tendons are frequently ruptured in falls and the long adductors of the thigh are frequently ruptured when 'doing the split'.

Water may enter body orifices under high pressure when a skier falls when moving at high speed. Sinusitis or otitis media is a frequent complication from water entering the nose or ears. Choking and coughing is produced by water entering the mouth. Damage has been done to the rectum and the vaginal cul-de-sac has been ruptured when a skier has fallen in a sitting position while travelling at high speed. Bathing caps are the best protection for the ears. Ear plugs may be forced further into the ear canal.

The tow rope can do severe damage either at the time of take off or after a fall in the water. The rope may flip about a hand, arm, leg or around the neck causing severe brush burns, dislocations, deep lacerations or avulsion of the skin and soft parts. Brachial plexus injuries and fractures of the clavicles have been seen. At times a skier may be dragged long distances and drownings have occurred.

Striking a solid object while water ski-ing may produce a wide variety of

severe injuries. Fractures of any type can occur when a skier misjudges his distance and collides with a wharf, dock, house boat, floating debris, a partially submerged log, a large rock or a stump barely showing out of the water. Fractures of the hands, wrists, ribs, femurs, legs and pelvis are frequently seen. At times compound fractures or severe lacerations can complicate the picture.

Injuries by other boats are not uncommon. For this reason a tall flag on a float is a wise precaution to protect a skier who has fallen in the water. He may be run over by a boat with severe head injuries. Fatal accidents have occurred. Traumatic amputations of the extremities or digits have occurred from contact with boat propellors.

In spite of precautions and safety rules a good number of drownings continue to take place each year. It is surprising the number of people who ski but can't swim. They don't seem to realize that the safety belt can slip loose or come off.

The American Water Ski Association publishes a magazine—The Water Skier—that appears each two months—six times a year. There are many articles and helpful suggestions on the prevention of injury. Attempts are being made to secure legislation so that standardized rules can be enforced. Police patrols or game wardens are presently enforcing many regulations.

SUGGESTED REGULATIONS

The understanding of a few simple requirements could be of great help in the intelligent prevention of many injuries. Don't ski at night. Don't ski when fatigued, cold or stiff. Do be in good condition for ski-ing and always wear a life belt.

There should be at least two persons in the boat, one to drive, the other to watch the skier.

A wide angle, rear view mirror will help the driver.

The driver should be experienced and responsible. He should keep his seat at all times and not take chances with sharp turns.

The boat should carry a flag to indicate that a skier is being towed. The American Water Ski Association has one for this purpose.

The skier should be a good swimmer or learn to swim.

(a) He should not ski in unknown or shallow water.

(b) He should never wrap or fasten the tow rope to any part of his body while ski-ing.

(c) He should be careful to see that the tow rope is clear before the take off and he should always watch the water ahead while ski-ing.

(d) He should be courteous and stay a safe distance from other skiers, swimmers, fishermen and other boats.

(e) He should stay clear of solid objects and should approach his dock or landing parallel with the shore line.

(f) If he falls he should avoid, if possible, the spreadeagle or forward position.

(g) When in the water he should hold up a ski to warn approaching boats or other skiers.

SUMMARY AND CONCLUSION

Water ski-ing has been presented as a pleasant sport that is becoming increasingly more popular.

The hazards of water ski-ing and the types of injuries that occur have been discussed. Injuries in water ski-ing are perhaps more frequent than is realized.

No attempt has been made to outline treatment. Fundamental principles of treatment are the same as for other similar injuries.

The author has long been of the opinion that, when possible, immediate surgery should be carried out in cases where surgery is indicated.

The need for a constructive educational programme and proper regulatory measures concerning water ski-ing is emphasized.

20

WINTER SPORTS

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THE INJURIES which result from winter sporting accidents have certain particular characteristics which distinguish them from other sporting injuries. Factors determining the type of injury sustained include the mechanical leverage of skis, the vulnerability of the unsupported human frame travelling at high speed, the hard frozen surfaces and the effect of cold and fatigue on muscular function.

High speeds and hard surfaces are the factors which influence injuries sustained in skating and tobogganing. These injuries are usually the result of striking ice, rock or other hard objects. The mechanism of injury is obvious and the severity dependent upon the force and the part injured. Head injury is the most important and is quite common. Protective clothing has been developed to protect the knees and elbows of skaters and the heads of those racing on sleds or toboggans.

Ski racing and jumping carry similar risks to those involved in tobogganing and the hazards are well appreciated by those expert skiers who compete in such sports. Fatal accidents which occur each year are usually the result of head injury: there would, therefore, seem to be good reason to advocate protective headgear: but it is probable that the racing skier would be even more reluctant than the racing motorist to hide his bravado beneath a crash helmet.

The injuries so far described affect only a small body of expert winter sportsmen and comprise a very minor part of the large problem of winter sporting accidents. The suntanned ski tyro with one leg in plaster is a common sight in many countries of the world and gives a true representation of the commonest winter sporting injury. Ski-ing is fast becoming the most popular of all participation sports and the injuries to which skiers are prone are usually characteristic and often preventable. It is estimated that three and a half million people ski in the United States alone and that injuries occur at the rate of 5 per 1,000 skiers per day (Erskine, 1959).

Table I summarises the incidence of injuries taken from four reports during the past decade. The great difference in the proportional distribution of

TABLE I

Distribution of Ski-ing Injuries

Author	Baumgartner	Morrison et al	Moritz	Williams
Venue	Bavaria	New England	Rocky Mts.	British troops in Austria
Site of injury	%	%	%	%
Head and neck	4·5	0		0
Upper limb	13·6	13		1·7
Trunk	3·4	4	(lower limb only)	0
Thigh	2·5	4		0
Knee	17·2	22	18·4	47·8
Lower leg	22·1	10	8·5	4·4
Ankle (strain)	20·4	20	54·9	40·7
Ankle (fracture)	13·1	25	16·9	5·3
Tendo-Achilles	0·7	0	1·3	0
Foot	2·5	1·5	0	0
No. of cases	1,000	240	461	113

injuries is due to a number of factors, the most important of which is the type of skier involved. In the series of Baumgartner (1960) a high proportion of skiers were experienced and probably skied each week-end while in the small series which I reported (Williams 1956) virtually all those at risk were novices.

During the last two decades a change in the incidence of certain injuries has been noted. Rupture of the tendo-Achilles which was extremely uncommon before 1940 has gradually become more common with the adoption of the firmer fixation of the heel to the ski (Jakob, 1957; Schonbauer, 1959; Moritz, 1959; Thomsen, 1959). With the improvement in ski boots and the increase in support which they impart to the ankles the incidence of fractures of the shaft of tibia and fibula has increased while that of ankle fracture has decreased (Bernbeck, 1958).

TYPES OF INJURY

Head and neck

High speed direct trauma is the most important cause of head injury while ski stick lacerations or puncture wounds are becoming less common with the adoption of metal poles which bend rather than break.

Cervical disc lesions are quite common particularly in the middle aged holiday skier when pre-existing degenerative disease is activated by unaccustomed minor trauma (Bengui, 1960).

Upper limb

Almost any type of injury may result from collision or lateral falls onto hard surfaces, but two injuries are quite common, dislocation of the shoulder and the 'ski pole fracture'.

Dislocation of the shoulder often occurs in falls during slalom racing when the outstretched hand becomes fixed in the soft snow as the body continues to descend the slope. The shoulder is abducted, extended and dislocated. Immediate reduction at the place of injury can often be accomplished and may save the victim an uncomfortable and possible expensive descent in a 'blood waggon'. It must be remembered, however, that fracture dislocations can occur in which attempted reduction may do more harm than good.

'Ski-pole fractures' occur in an outstretched hand which is holding a ski stick. The metacarpals are usually affected, either a fracture of the third and fourth (Morrison and Coughlin, 1960) or more commonly an adduction injury of the first metacarpo-phalangeal joint (Weller, 1961).

Trunk

The commonest trunk injury is the aggravation of an existing lumbar disc lesion (Baumgartner, 1961). As with cervical disc injury this is more likely to occur in the older skier returning to the snows for two weeks strenuous activity after spending fifty weeks in a sedentary occupation. Other trunk injuries are usually the result of collision.

Thigh

Soft tissue injuries and fractures of the thigh are not very common and usually occur as a result of direct trauma such as collision or striking a rock or tree (Cozzolino, 1957). A lateral blow may result in a fracture in the region of the trochanters (Morrison and Coughlin, 1950). Fractures as a result of torsion strain are rare in the femur.

Knee

The knee joint is second only to the ankle in its susceptibility to ski-ing injuries. The injuring force is abduction, but there is also some rotation. 56·4 per cent of all torsion injuries to the lower limb result in a knee injury, and of these 29·4 per cent are associated with an ankle strain and 7·9 per cent with an injury to the opposite knee. The medial collateral ligament is damaged in 87·1 per cent, the lateral in 1·4 per cent and the cruciate ligaments in

only 0·8 per cent (Reissigl, 1955). Medial meniscus injuries are uncommon (Reissigl, 1955; Watts, 1956; Williams, 1956; Bätzner, 1957). Complete rupture of the collateral and cruciate ligaments are rare, but this injury must always be excluded, if necessary by examination under an anaesthetic. Prompt surgical repair of completely ruptured ligaments is essential (O'Donoghue, 1959).

The common knee injury is a partial tear of the deep fibres of the medial collateral ligament between their attachments to the femur and the meniscus. Figure 1 shows the site of injury and the direction taken by the consequent haematoma. The complication of naemarthrosis occurs in 5 per cent of these

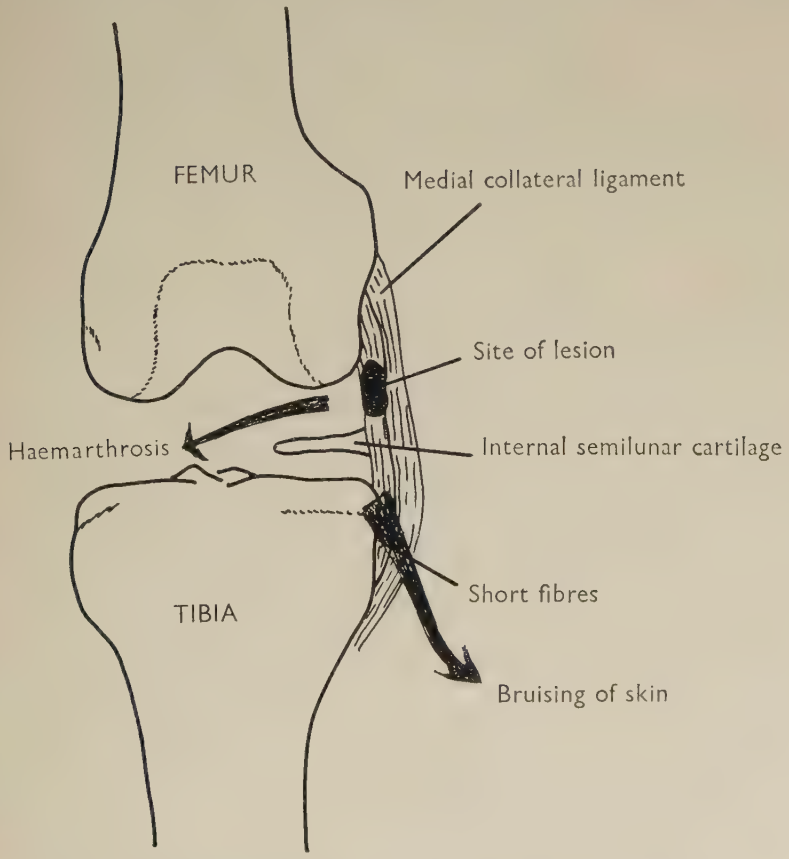

FIG. 1. Diagram showing the usual site of rupture of the medial collateral ligament and of the associated bruising of the skin.

injuries and is suggested by a very rapid onset of effusion, by local heat and pyrexia. Extravasation of the haematoma downwards results in the appearance of a bruise and often persistent tenderness well below the actual site of injury (Williams, 1956).

Lower leg

The only common form of direct trauma is a laceration from the edge of the opposite ski. By far the commonest injury is a fracture of both leg bones due to a rotational force. Erskine (1959) suggests that the greater the force the higher in the leg the fracture occurs. The fracture of the tibia is commonly in the lower half, spiral and often producing loose 'butterfly' fragments. These factors tend to make the fracture unstable and increase the risk of delayed union. There is considerable difference of opinion concerning the best method of treatment of these fractures. The desirability of early transport of the patient away from the ski-resort is an important part of the argument against prolonged skeletal traction. Open reduction is commonly employed, but the continental practice of circumferential bone wiring (Jantzen, 1959) is not favoured by British or United States surgeons, who prefer screw fixation, intramedullary nailing or primary bone grafting.

Ankle

Ankle injuries are almost invariable rotational. In the unsupported lower limb a rotation force applied to the foot will usually produce an injury at the ankle joint. If the force is applied slowly the ligaments are likely to tear first and if the force is applied rapidly the bones of the ankle mortice are more likely to fracture. This is not an invariable rule and the result depends upon the relative strength of bone and ligament. Approximately half the ankle injuries seen are fractures and half are strains but in novices strains predominate. Internal rotation injuries are commoner than external and plantar-flexion injuries almost never occur. Any of the ligaments guarding the talo-tibio-fibular joint may be involved. Complete rupture is relatively uncommon but must always be suspected and examination with X-ray under anaesthesia is often necessary. The advantages of examination early after the accident in order to assess the site and severity of the injury are stressed by Moritz (1959). In a fracture of the ankle it is particularly important to know whether the joint is involved or whether diastasis has occurred, as these factors greatly influence treatment and prognosis.

Tendo-Achilles

Subcutaneous rupture of the tendo-Achilles just above the level of the boot is becoming a more common injury and is of great importance because of the

disability which may follow incorrect treatment. Unless the lesion is suspected it is possible for the diagnosis to be missed. (Jakob, 1957).

Foot

Injuries to the foot are made up of a collection of many different lesions, from lacerations and punctures to rotational and fatigue fractures. No one injury is particularly common.

Mechanism of injury

Direct trauma is the cause of approximately one fifth of all ski-ing injuries. Rocks, trees, other skiers, the opposite ski or the sticks usually inflict the trauma. The injuries which result have no particular characteristics with the exception of the rather rare puncture wounds due to ski sticks. The large majority of injuries are the result of some mechanism peculiar to the strains of ski-ing, or to falling into deep snow. The common upper limb injuries are due to falling on to an outstretched hand holding a stick or to the arm getting fixed in deep snow. If the skier held his arms into the side when he fell in deep snow, he would run far less risk of injury. The instinctive response, however, can only be overcome by training and practice.

Lower limb injuries are predominantly rotational injuries. In novices the common mechanisms of injury are crossing the skis on a ski-tow or while attempting a 'snow plough' manoeuvre or divergence of the skis when traversing or running into deep snow. With the more experienced skier the commonest cause is catching an outside edge when turning or an inside edge when traversing. The ski then becomes fixed in the snow while the body continues at the same speed. The momentum of a man ski-ing at 30 m.p.h. is 6,600 ft. lbs. per second and as the leverage of the skis creates a moment, the resultant rotational force is very great (Erskine, 1959). There are two movements which come into play if the ski tip is fixed, one between the tip and the boot and the other between the centre of gravity of the body and the ankle. The greatest force is applied at the ankle but if this is well supported by a rigid boot the force is transmitted through the tibia and fibula to the knee. If the force is rapidly applied as in a fall at speed, fracture of the rigid bone occurs. If the force is more gradually applied as in the slower falls of the novice the bone does not fracture but transmits the force to the ligaments. This explains why ligament injuries of the ankle and knee are much more common in the slow inexperienced skier.

If the fall is produced by the ski tip catching in an obstacle during a direct downhill run, the body moves forwards on the ankle and a tremendous dorsi-

FIG. 2. Injury usually only occurs in unskilled performers. High speed competitive ski-ing does, however, entail a certain amount of risk.

flexion force is exerted on the ankle. If the boot is firmly fixed to the ski, as in most modern bindings, a tendo-Achilles or peroneal tear may occur. If the ski tips are less firmly fixed in the snow the resulting fall is picturesquely described as an 'egg-beater'. In such a fall the traumatising forces are dissipated over time and distance and the skier may escape injury. However, if the skis cross, rotational injuries may result. In addition such injuries as upper limb dislocations or 'ski pole' fractures are possible (Erskine, 1959).

The prevention, or minimisation, of these characteristic lower limb rotation and ankle dorsiflexion injuries is the purpose behind the development of release bindings. The commonest type of release binding is designed so that the toe is freed from the ski if a sudden rotation force is applied. A compromise must be reached between a binding which will not be operated by the considerable forces developed during sharp turns and one which will operate before sufficient force is transmitted to ankle or ligament to cause injury in a fall. The number and variation of available release bindings is an indication that the perfect 'safety' binding has not yet been developed. The selection and

adjustment of release bindings is a subject on which expert advice is necessary: and the reports of the various agencies, such as the Ski Club of Great Britain (Young, 1961) which have studied the problem, is of great value. Although injuries may not always be prevented, the wearer of correctly adjusted safety bindings is less likely to injury than the unprotected skier and, if injured, is likely to sustain a less serious injury (Howkins, 1958).

The increase in incidence of tendo-Achilles tears has led to the development of a number of bindings which release if the skier falls forward (Hentschel, 1959). The majority of these are a separate device and not part of the, now common, rotation release bindings.

Important predisposing factors in the production of injury are inexperience, loss of confidence and fatigue, all leading to loss of ski control. There are no short cuts to experience: but good tuition ensures that it is acquired as painlessly as possible and put to the best advantage. Loss of confidence is closely related to both inexperience and fatigue, and often results from unexpected changes in snow conditions (Truchet, 1957). Loss of confidence following injury is an important factor in predisposing to further injury. Kraus (1959) reported that 40 per cent of the knee injuries and 14·2 per cent of all injuries in a large series were re-injuries. The gradual rehabilitation after injury is therefore important, and skiers would be advised to rejoin ski classes of a lower standard than the one they reached at the time of injury.

Fatigue is perhaps the most important single predisposing factor, and this can be combatted in part by appropriate pre-ski exercises, and dry ski-ing or by knowing when to stop ski-ing. For most participants, ski-ing is more a sensual pleasure than an athletic pursuit, and however much they are exhorted to sway on half bent knees for a month before their holiday, or to spurn ski lifts for the first five days, the majority will make their first journey of the year on snow downhill from the top of a ski lift using untrained muscles. It is well known by all concerned with the transportation and reception of injured skiers that peak accident times are just prior to lunch and at the end of the day, (Erskine, 1959; Moritz, 1959 and Staples, 1961). It would seem, therefore, that one of the greatest dangers in ski-ing is taking 'just one more run down', and that perhaps the greatest advantage of spurning ski lifts is that it is a good deterrent from taking another run when tired at the end of the day.

In my opinion the most important steps to be taken by a skier in the prevention of injury are the acquisition of correctly adjusted release bindings which release with forward as well as rotational strain; to spend more money on ski tuition and less on lifts; and, most important of all, to act maturely, particularly at the end of the ski-ing day, and resist the temptation of the last run of the day.

REFERENCES

Batzner, K. (1957). *Dtsch. med. Wschr.*, 82: 276.
Baumgartner, W. (1960). *Munch. med. Wschr.*, 102:2220.
Baumgartner, W. (1961). *Ibid.*, 103:507.
Bengui, A. (1960). *Rev. Rhum.* 27:274.
Bernbeck, R. (1958). *Munch. med. Wschr.*, 100, 547.
Cozzolino, A. (1957). *Arch. Ortop.*, Milano. 70, 133.
Erskine, L. A. (1959). *Amer. J. Surg.*, 97, 667.
Gruenagel, H. H. and Adloff, D. (1963). *Dtsch. Med. Wschr.*, 88, 711.
Hentschel, M. (1959). *Munch. med. Wschr.*, 101, 2408.
Howkins, J. (1958). *Brit. med. J.*, i: 1006.
Jakob, F. (1957). *Minerva Ortop.*, 8: 216.
Jantzen, P. M. (1959). *Munch. med. Wschr.*, 102, 718.
Karvonen, M. J., Rautanen, Y., Rikkonen, P. and Kihlberg, J. (1958). *Ann. Med. int. Fenn.*, 47:75.
Kraus, H. (1959). *J. Amer. med. Ass.*, 169, 1414.
Moritz, J. R. (1959) *Amer. J. Surg.*, 98:493.
Morrison, G. M. and Coughlin, E. J. (1950). *Ibid.*, 80:630.
O'Donoghue, D. H. (1959). *J. Amer. med. Ass.*, 169, 1423.
Reissigl, H. (1955). *Dtsch. med. Wschr.*, 80, 183.
Staples, O. S. (1961). *New. Engl. J. Med.*, 264: 552.
Truchet, P. (1957). *Minerva Ortop.*, 8: 236.
Tucker, W. E. (1957). *Practitioner*, 179:1687.
Watts, J. P. (1956). *J. Roy. Army Med. Corps.*, 102, 48.
Williams, J. A. (1956). *Lancet*, i, 96.
Young, N. J. E. (1961). *British Ski Year Book.* Ski Club of Great Britain.

21

SKI-ING

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STATISTICS from travel agencies indicate that 100,000 people from Britain ski, and probably half this number go out for a fortnight's holiday from otherwise sedentary occupations, unfit and untrained. Considering the number, and the circumstances, it is not surprising that the annual total of accidents appears large, and tends to earn this sport the reputation of being dangerous. The veterans who have skied for twenty-thirty years or more claim that this never used to be so, and tend to throw the blame on modern ski-bindings and techniques. I feel it would be more accurate to lay the responsibility on the vast increase of uphill mechanical transport since the second war. Nowadays, instead of climbing up every inch of the way to have about an hour's downhill ski-ing, in the day, thousands of skiers are discharged each hour by trains and ski tows, 'un-warmed up', onto very high and difficult descent courses which previously were only accessible to the very experienced. The tyro today may achieve five or six hours downhill ski-ing by these means, thereby tiring untrained and otherwise unused muscles, until they cease to serve their purpose. Falls occur sometimes at considerable speed, and the leverage exercised by skis during a tumble damage the limbs to which they are attached.

The most common accidents are therefore to the legs. Perhaps five per cent of the total are attributable to the direct impact of any part of the body onto hardy objects on the ground, such as rocks, hard ice, etc., and take the form common to any other incident of this sort—concussion and dislocation of the bones of the upper limbs. However these are few compared with the leg injuries.

Injury to the lower limbs varies in severity from torn muscles and tendons to dislocations and fractures, according to the speed and the degree of relative motion between body and skis. When the snow is soft, or deep and soggy, the skis become held relative to body movement more than on the polished tracks of the much used 'pistes'—when all tend to slide on together. The pistes carry 90 per cent of the traffic however, and it would be wrong to de-

duce that 'off the piste' is the scene of the largest number of accidents. This is not the case for two reasons: Firstly it requires more and better technique to operate off the piste; secondly, speeds off the piste are much lower. But the piste skier out of control, for countless reasons, may run off the track into the rough at the sides at high speed; and the sudden change of terrain causes the fall that might otherwise have been averted.

Ski racers are a case apart. They accept calculated risks and their speeds are very great. They do not really come into this study, except insofar as the injuries fall within the usual pattern.

In a report to the Ski Club of Great Britain on ski-ing accidents which involved fractures among British holiday skiers at one centre over a period of five years, the summary read as follows:—

(1) 80 per cent of leg damage is the result of 'rotation' of the skis relative to the limb.

(2) About 5 per cent can be attributed to the purely forward fall, where the ski is retained and the skier goes on—and the damage is usually to the achilles tendon.

(3) 10 per cent is caused by impact of one ski upon the other shin, and

(4) the remainder is due to direct impact with hard objects.

What is being done to reduce the accident rate? In recent years a great deal of effort and ingenuity have been devoted to developing 'Release Bindings', the binding being the attachment of the boot to the ski. The principle is to hold the boot rigid on the ski (so that the wearer can turn the skis), but only up to a certain pressure, lower than that at which limb damage may occur. If this pressure is exceeded the bindings become unfastened from the boot and relieve the strain on the foot. The idea is excellent but the selected pressure is always a compromise. Fairly heavy pressures are sometimes necessary to drive the ski in deep snow and in the brusque turns demanded by the slalom.

I myself believe that ignorance is to blame for most accidents; release bindings are useless unless the skier knows how to set them and maintain them; good equipment is wasted if the skier does not take the trouble to learn the basic techniques; and expert technique is no guarantee of safety if the skier has not the sense to prepare his muscles for the season and warm them up before a run.

The chief hope then of reducing the accident rate is education. By energetic propaganda and good teaching, tasks for which ski clubs and ski schools are the best agents, it is possible to cut down accidents by more than half. The ski centre at which the review quoted above was made, has, by educating, reduced the casualty rate during the last three seasons by two-thirds.

PART III

Section I: Soft Tissue Injuries

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C.V.O., M.B.E., T.D., F.R.C.S.

1

THE DIAGNOSIS OF SOFT TISSUE AND JOINT INJURIES

EVERY athletic injury is a soft tissue injury regardless of other structures which may be involved. What proportion is of soft tissue alone is difficult to estimate. At Harvard University, where all injuries are recorded and classified, sprains, strains and contusions account for two-thirds of the total (Thorndike, 1959).[1] Simple injuries of this sort may lead to a great deal of disability, loss of function, and wasted time; it is important to educate and encourage the athlete to take them seriously. If he is to get fit quickly he must have the benefit of immediate and effective treatment. Only too often a soft tissue injury is considered trivial and treated inadequately.

Important anatomical and physiological factors connected with diagnosis

A knowledge of the following anatomical and physiological factors is important as to the understanding of the pathological conditions and as an aid to diagnosis.

Fascial planes: A knowledge of the extent and direction of the various fascial planes will help in appreciating the wide range of spread of the products of haematoma formation. For example, the spread from a haematoma over the subcutaneous border of the tibia just medial to the tibial tuberosity will be down the shin and medial side of the calf to near the ankle joint. If all the products have not been absorbed by the time they reach the convergence of the fascial planes about three inches above the medial malleolus a puffy swelling will form here.

Extensibility and flexibility of muscles: Many muscles exert their function on more than one joint and in many cases over several. For instance, the superficial flexors and extensors of the fingers proceed from their origin above the elbow, down the forearm, across the wrist and carpus to the fingers. The full extensibility of the finger flexors is when the elbow is extended and the wrist fully dorsiflexed, either fully supinated or pronated with the fingers

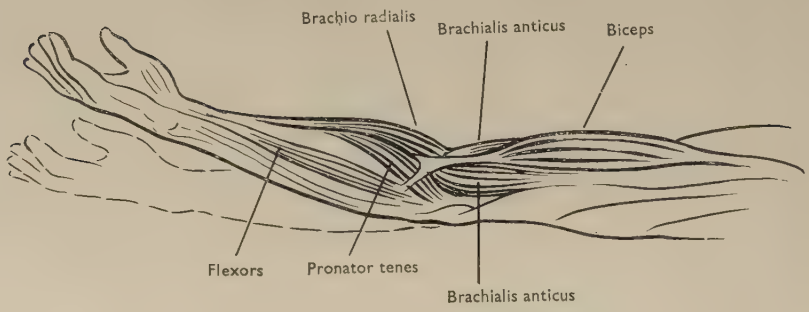

FIG. 1. This illustrates how contraction of the biceps, brachialis anticus, forearm flexors and pronators can prevent full extension of the elbow joint and dorsi-flexion of the wrist joint.

extended. The extensibility of the extensors is when the elbow is fully extended, the wrist palmar flexed and the fingers making a full fist. The extensibility of a muscle is often limited by either adhesions or thickenings in a muscle, or by muscle spasm. The full flexibility of a muscle may be limited by muscular adhesions or spasm but is usually due to capsular or intracapsular causes. Full function of a muscle or joint is unlikely unless both have full extensibility and flexibility.

Bursae: Bursal sacs are placed usually between a muscle or tendon working over a bony point, for example the large bursa under the gluteus maximus. The amount of synovial fluid in each bursa varies considerably but as the deep surface of the bursa is fixed during both contraction and relaxation of the muscle, the fluid content of the bursa allows for compression and dilatation on varying parts of its superficial surface. As a muscle contracts, so one part of the bursa will be flattened and the other distended.[2] This change of the tension in different parts of the bursa can be likened to the flow of protoplasm in an amoeba. Any disturbance in the normal rhythmic action of the overlying muscle is liable to stimulate the synovial membrane of the bursa and produce a bursitis.

Blood vessels: The position of the various blood vessels in relation to an injury must be given careful consideration. In many situations, especially in relation to the knee and elbow, there is a fine network of blood vessels extending around every aspect of the joint. Haematoma formation associated with periostal stripping often will lead to ossification either in the capsule or the joint or surrounding muscles or tendons especially if the part is moved excessively or given too vigorous treatment.

There would appear to be a connection between the subperiostal blood vessels of the surface of the femur with those in contact with the deep surfaces of the quadriceps, adductors, and hamstrings.[3] Rupture of these vessels will lead to the formation of a deep seated haematoma liable to calcification or myositis ossificans.

A knowledge of the position of the superficial blood vessels and cutaneous nerves is helpful as thickenings due to consolidation of traumatic swelling often form at these points and are a persistent cause of pain. In the lumbosacral region painful fatty lipomata may appear where the blood vessels and cutaneous nerves pierce the deep fascia.

The joint lubricating system: Our knowledge of the mechanism of joint lubrication is still incomplete.[4] Synovial fluid is a viscid, slippery fluid derived by dialysis from plasma enriched by polysaccharides including hyaluronic acid.

There are three theories of how joints are lubricated:—

1. The fluid system of Barnett, Davies and MacConaill.[5]
2. The boundary lubrication mechanism of Charnley.[6]
3. The weeping system of McCutchen and Lewis.[7]

Barnett, Davies, and MacConaill suggest that the fluid acts as the liquid component of a fluid film system. Their explanation of this system is one in which a lubricant coats the moving surfaces and remains an integral part of them while the actual movement occurs between other parts of the fluid. Articular cartilage can be shown to imbibe and expel synovial fluid as the pressure on it varies.

Charnley gives 0·01-0·02 as the friction coefficient of the human ankle joint and demonstrates this low friction is not due to hydrodynamic lubrication. He postulates that a boundary lubricant is one which will operate 'between solid surfaces separated by a fluid film which is too thin to behave as a fluid . . . it reduces friction by entering into physiochemical combination with the surfaces, and diminishes the free molecular attraction which may seize the surfaces together.'

McCutchen and Lewis put forward a theory which can account for low friction lubrication. They suggest that articular cartilage is ideal for absorption of synovial fluid which when pressed on by its opposing articular surface exudes synovial fluid in a manner which they call the weeping lubrication of joints.

Articular cartilage resembles a rather stiff sponge with an internal structure which permits easy expression of fluid to a smooth porous outer surface. Thus when an individual is resting, the articular cartilage is absorbing syno-

vial fluid which is squeezed out slowly to give what is called the weeping lubrication of joints.

Their proof for this is:—

(a) Structural. Benninghof's description of the arrangement of cartilage cells would allow for the essential structure of such a sponge.

(b) Photometer Analysis by a flame photometer: articular cartilage can be shown to contain a high proportion of extra cellular fluid. Exudation of only a small fraction of this would provide an adequate lubricating film.

Although it has not yet been proved possible to devise a crucial experiment to prove conclusively this hypothesis, all present evidence suggests that this type of lubrication does in fact occur.

Diagnosis

Accurate, early diagnosis is essential for proper treatment. It is sometimes impossible to make more than a generalisation at first, however careful and exhaustive the investigation may be. For example, an injury to the knee joint may have caused a sprain to the medial collateral ligament locally, and a strain of all the structures of the joint generally. It may not be evident at the earliest stages whether other structures, such as the medial meniscus or cruciate ligaments, are also damaged.

As treatment proceeds and the patient is seen from day to day, a more definite diagnosis is possible; so that measures can be directed towards specific as well as the general lesion.

The history

To arrive at a diagnosis, one must take a detailed history, not only of the present accident, but of any previous injuries. The injured part must be examined carefully, not in isolation, but as part of an anatomical segment, and then as part of the body as a whole; for there may be some associated injuries or some general or segmental spinal condition exerting a secondary effect.

The following questions will help to establish the diagnosis:—

Nature of the injury: How did the injury occur, and what was the position of the injured part before, during and after the accident? Many of the common athletic injuries happen during a particular movement when the joint is in a particular position. It is essential to have a clear conception of the mechanics of the injury; was it pure abduction or adduction strain, or was there a degree of flexion, hyperextension or rotation as well?

Type of onset: Was the onset gradual or sudden?

Pain: Is the part painful? If so, is the pain continuous or momentary?

Does it occur at a particular time of the day or night? Is the pain present at rest or only during movement? Ask patient to localise point of minimum pain by pointing with one finger. It is important to distinguish between a simple muscle injury and one which involves the associated joint. In a muscle strain or tear, the area of tenderness is localised to the injured muscle and active movement is painful. Passive stretching of the muscle may also produce pain and the muscle may go into reactionary spasm.

When the joint is involved the tenderness is usually more diffuse unless a ligament only is torn; movement is painful and there is often a painful arc of movement. There may also be protective spasm of a group of muscles if forced movements are attempted.

At this point it is appropriate to consider the interpretation of pain, both by the surgeon and by the patient. Pain may be due to local causes, such as an area of inflammation, which is invariably very tender to touch; or it may be referred. An inflamed viscus causes pain in the region supplied by the somatic elements of the visceral nerve. Diaphragmatic irritation from either a basal pneumonia or subphrenic abscess for example frequently produces pain in the shoulder; displaced nuclear fragments from a lower lumbar disc may come into contact with the root of the sciatic nerve and give rise to sciatica; hip injury or disease may cause referred pain in the knee joint.

During the last twenty-five years, intervertebral disc lesions have become widely recognised and, as a result, pain of local origin is often mistakenly ascribed to the neck or the back. The injection of local anaesthetic (3-5 ml. of 2 per cent procaine or xylocaine) into the tender area can sometimes help to establish whether pain is local or referred.

Swelling: Is there any swelling or deformity of the part? And did it occur gradually or suddenly?

Movements: Do certain movements appear restricted? Often in joint injuries, only the extremes of movement are limited, and the patient does not appreciate the restrictions until he has been asked to produce the full range of active movement. Where the knee is concerned for example, the normal patient should be able to make a full knee bend standing on the tip of his toes.

Locking: Has the joint locked? It is important to ascertain from the patient the distinction between ordinary stiffness, muscular spasm as the result of intra-articular inflammation and a momentary catch. The true locking which blocks further movement may occur at a certain point, at which the joint becomes fixed and then if it unlocks, how does this take place? As a criterion of true locking the patient must be able to describe a manoeuvre he carries out himself to unlock the joint.

Crepitus: Is the patient conscious of creaking, grating or snapping of certain soft tissue structures over bony points? Are these felt or are these heard? Can he describe at what level or depth the derangement occurs?

Stiffness: Does the injured part feel stiff after rest or early in the morning? or does the stiffness come on when he has been on it all, or some part of the day? What is the reaction to exercise or to any previous treatment that may have been given?

Stability: Does the part feel insecure, and, if it is the lower limb, does it tend to give way on movement whilst weight bearing?

Temperature: Does the injured part feel hot or cold? The temperature of the skin over a tender point is often several degrees higher than that of the surrounding area, and the difference can be appreciated by the examining fingers.

Circulatory disturbances: Does the part distal to the injury feel numb or tingle? or does the circulation in any way feel different? That is to say does the area ever become pale and cold, or flushed and hot? Does it perspire?

No history would be complete without an enquiry into the previous health and the family history of the patient. Information on the following points may be important:—

congenital abnormalities; previous injuries; focal infections; acute or chronic sepsis; rheumatic conditions; metabolic disturbances; malignant disease.

A family history of an inherited abnormality may be especially important in the diagnosis of an atypical condition.

Answers to many of the questions asked the patient may be confirmed during clinical examination. If the patient is unconscious or unable to co-operate, the surgeon must rely on the clinical examination, his own clinical experience and judgement, and reports from eye witnesses.

Examination

During examination special attention should be directed to the following physical features:—

General: The patient's appearance and apparent state of health. The stance of the patient and his gait.

Local: The position in which the injured part is held and its temperature (whether hot or cold) compared to the other side. If it is swollen or oedematous: what is the nature of the swelling—muscular, synovial, cartilaginous or bony? Is it uniform, fluctuant, tense, tender, translucent, symmetrical or does it pit on pressure?

The circumference measurements of the two limbs at several levels should be compared.

The presence of a tender point or points: One can usually locate tender points accurately by moving the examining finger lightly over the injured area until one comes upon the point of maximum tenderness. It is important to appreciate the different feel of certain thickenings in the tissues and also the different degrees of tissue tension. In early cases of chrondromalacia patellae, for example, the infrapatellar pads of fat often become tense and tender to palpation. It is also possible, to appreciate differences in the thickening in a joint capsule or tendon sheath. Stenosing tenovaginitis, for example, can be detected at an early stage. Often, too, a group of muscles may be strained or contused and if the antagonistic group is examined they will be found to be tender. Tenderness is frequently found in the substance of the antagonistic group as a result of imbalanced muscle action. An example of this is a sprain, contusion or tear of one of the adductor muscles of the thigh. These, apart from adducting the thighs, externally rotate the limb. If the corresponding gluteus medius and minimus are examined they are often found to be tender, and internal rotation may be limited. Another example is the tenderness of the flexors and pronators of the forearm in an established case of tennis elbow where the typical tenderness of the tissues is localized to the extensor origin and supinator brevis over the outer side of the elbow joint.

Examination for muscle involvement: It must be remembered that some muscles traverse and move more than one joint; and, therefore, the movements of the joints above and below the injured structure must be examined as a routine.

One must assess the state, tone and feel of the muscles in the vicinity. By experience one can assess by touch the tone of muscles. Often the feel of atonic muscles can be a more reliable test than the actual comparison of the circumferential measurements of the limbs. The feel of normal relaxed muscles is easy to appreciate. The normal muscle feels resilient, has no tension or toughness, and has a normal response to faradic stimulation. Compare the tone and feel of the muscles in the contralateral, normal limb with those of the injured side.

Muscles affected by pathological conditions often have a characteristic feel. Muscles in spasm, for example, are firm, often painful on stretching and sometimes tender. This tension appears to be quite different from the tension of muscles affected by postural or occupational strain which often feel like old rubber and are tense and tender; or muscles in which there is a true fibrotic state, following trauma or neurological disease. These feel hard, even board-like and have lost their elasticity. They may not even be tender.

Examination for joint involvement: Exactly how much voluntary and involuntary movement is there? It must be remembered that in addition to its range of voluntary movement, each joint has a range of involuntary movement which allows for gliding, rotation and twisting of the bones one upon the other. The involuntary range must be full before there can be full range of voluntary or active movement. Movements against moderate resistance in every direction should always be tested to detect weakness of the supporting muscles. A thud or a click during the movements of a joint suggest an internal derangement.

Is the joint stable or unstable? What is the degree of damage to the ligaments? Is there any locking? Observe the adjoining muscles to see whether they are thrown into spasm.

Examination of bone, vascular and nerve injury: Complications such as damage to the underlying bone or the main blood vessels and nerves, either motor or sensory, must be excluded. One must check the pulse distal to the site of the injury, and examine for loss of muscle power and sensation, and for any vasomotor or trophic disturbances.

Special investigations

It is often necessary to carry out special investigations in order to confirm a diagnosis. For example, some radiographic investigations are not without danger when repeated; before ordering them one must weigh the advantages and the disadvantages:—

1. Radiological examination: Radiographs should always be taken in at least two planes if there is the slightest suspicion of bone or joint injury. They may reveal an unsuspected fracture, the presence of calcarious deposits, undue laxity of a joint producing a subluxation or some abnormality such as spondylolisthesis. One is apt to assume that a normal radiograph signifies normal anatomy, but one should always interpret the plates in association with the history and the clinical signs. The damage to the soft tissues of a joint may be evaluated more precisely by the aid of radiographs taken in several planes with tension stresses applied to the joint structures in different directions.

2. Special radiological examination:

(a) Arthrograms: these are techniques for outlining the joint structures. Under an anaesthetic filtered air may be injected into the knee joint for example. Somerville[8] has published some very important data on the results of such arthrograms. Similarly the shoulder and hips may be examined with the aid of opaque substances introduced into the joints.

(b) Myelograms: in a few spinal conditions, a myelogram is essential in

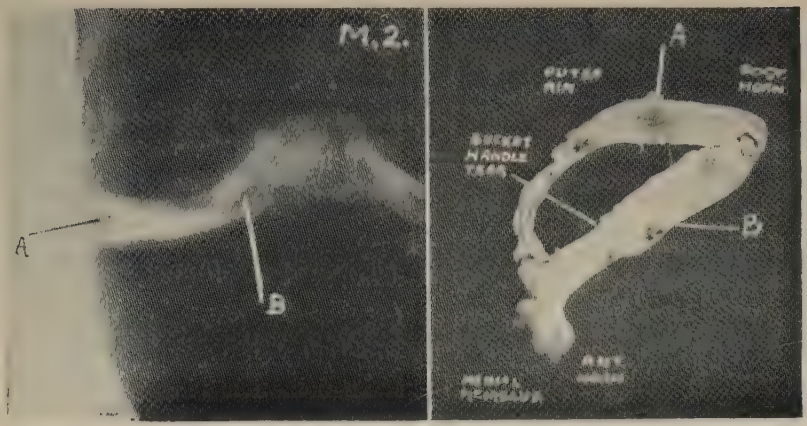

FIG. 3. A. Arthrogram showing bucket-handle lesion of medial meniscus: (A) outer rim; (B) Displaced bucket handle. B. Shows meniscus subsequently excised.
(From E. W. Somerville; *Journal of Bone and Joint Surgery*, Vol. 28, No. 3.)

order to demonstrate a space occupying lesion within or impinging on the spinal canal.

(c) Discograms: it is possible to inject normal saline or an opaque substance into the intervertebral disc for the purpose of demonstrating the possible presence of disc degeneration. However, this procedure is considered by many authorities to be unnecessary and dangerous.

(d) Arteriograms: in special neurological centres arteriograms play an important part in investigation of severe cerebral accidents which occasionally occur on the field of sport.

Electrodiagnosis: Until a few years ago routine electrodiagnosis was performed by the faradic-galvanic test. It is still sometimes used, but it is obsolete and is frequently fallacious. The faradic-galvanic test has been replaced by the Intensity Duration Curve, and the electromyograph.

The Intensity Duration Curve is a graph which is plotted to show the response of a muscle to an electrical stimulus of known intensity for a given time, which is produced by an electronic stimulator. The muscle to be examined is stimulated by the current passing through the muscle between two surface electrodes for a known time varying from 300m/secs. through 10 settings down to 0·1m/sec. The intensity of the current measured in volts or amps is noted and which produces a minimal contraction of the muscle for each set duration of time. A graph is then plotted of intensity of current against duration of current, and this is known as the Intensity Duration

Curve. From this graph it can be clearly stated whether a muscle is innervated or denervated or whether it is partially denervated. It is used extensively in following the recovery of muscles in peripheral nerve injuries (See Fig. 4, p. 334).

An electromyograph consists basically of a concentric needle electrode, an amplifier, and a cathode ray screen. The needle electrode is inserted into the muscle to be examined. The electrical potentials of the muscle are picked up by the needle electrode, amplified, demonstrated on the cathode screen, and heard on a loud speaker. From the nature of the wave patterns on the screen it can be determined whether or not the muscle is normal.

If an abnormal response is obtained it can be determined whether the lesion is a neuropathy or a myopathy. If a neuropathy, it can be determined whether the lesion is in the anterior horn cells of the motor nerve, in the axon of the nerve or at the myo-neural junction.

Examination of a joint under full general anaesthetic : Muscular spasm may be due to injury to an intra-articular structure such as a meniscus or an intervertebral disc, or to damage to the articular cartilage itself. It can be made to disappear during anaesthesia; it tends to return as soon as the patient regains consciousness, unless the derangement has been corrected. This sequence of events is not uncommon when a knee joint has been manipulated to reduce a torn meniscus. As it is often impossible to replace the torn meniscus, the physical signs and symptoms present themselves again very shortly after consciousness has returned. Some authorities take the opposite view; they consider that torn menisci and protruded discs can nearly always be restored to their normal position. At open operation, however, the surgeon is often able to demonstrate that the intra-articular structures are so damaged that it is impossible for any manipulative procedure to replace them into their normal position.

Adhesions on the other hand can be broken by manipulation under a general anaesthetic, and full movements of the joint return if there is adequate after care. Another indication for examination under full anaesthesia is in those cases of severe joint injury in which there may be a rupture of an important ligament. After a severe adduction sprain of the ankle joint for instance, there may be excessive movement when an inversion force is applied to the outer side of the ankle joint, producing a tilting of the talus. If an examination under anaesthesia is to be carried out for a suspected rupture of the lateral collateral ligament, the patient should be prepared for the operation. If the radiograph shows marked opening of the joint, operation can be performed immediately. The same applies to a suspected rupture of the medial collateral ligament of the knee joint.

If the examination is made some days after the injury, when some of the traumatic effusion has been absorbed, it may be possible to show some degree of laxity of the joint without anaesthesia. But if there is any doubt, the manipulation should be performed under full anaesthesia. In selected cases local anaesthesia may be helpful.

Laboratory tests: If the nature of the swelling, for example, is in doubt, the pathologist and the biochemist can give most valuable aid. Examination of the blood may indicate the presence of a rheumatic or gouty diathesis, infection or loss of blood; examination of fluid from a joint or bursa may reveal pathogenic bacteria; and examination of biopsy material may show a chronic infective or neoplastic process.

REFERENCES

1. Thorndike, A. 'Frequency and nature of sports injuries', Symposium on Sports Injuries. *American Journal of Surgery*, September 1959, p. 317.
2. McCutchen, C. W. and Lewis, P. R. Personal communication.
3. Crock, H. V. Personal communication, 1961.
4. *Lancet* leader 'Lubrication of joints", September 23rd, 1961, p. 700.
5. Barnett, C. H., Davies D. V. and MacConaill, M. A. 1961. 'Synovial Joints', Longmans, Green & Co., London, p. 304.
6. Charnley, J. 1961. *Lancet*, i., 1129.
7. McCutchen, C. W. and Lewis, P. R. 1959. *Nature*, Oct., vol. 184, p. 1285.
8. Somerville, E. W. 1946. *Journal of Bone & Joint Surgery*, Vol. 28, No. 3, p. 451.

2

PATHOLOGY

PATHOLOGY today focuses less on the postmortem room and more on the study of disease in the living body. It concerns itself, moreover, not only with morphological changes, whether macroscopic or microscopic, but also in the histochemical changes, early and late, in cells and tissue fluids.

Surgery has always been built on a thorough foundation of anatomy, physiology and pathology. The surgeon of the future, though he may not have to undergo training as a histochemist, must be aware that early histochemical changes occur before the more obvious structural ones; consequently he should be able to understand their significance.

The pathology of athletic injury differs in no way from injury in other spheres of life, and treatment should be governed by the same basic principles. It is one of the fundamentals of pathology that when a structure is damaged, nature tries to restore it as exactly as possible, but, owing to the variation of the tissues and of the ability of the cells in different areas to replace themselves when destroyed, the effort often fails. In most cases the best repair that can be expected is a supple, resilient, fibrous tissue scar.

Injuries occur as the result of:—

1. Direct trauma
2. Indirect trauma
3. Postural and occupational strain

These are the three main causes but one should always keep in mind the following subsidiary causes. Under this heading can be included:—

(a) heat, cold and draughts.

(b) vascular changes (which may themselves result from injury), arteriosclerotic or vaso-motor disturbances and occlusion of a vessel by a thrombus or embolus.

(c) infective causes. The part played by bacteria and viruses must be considered. There may be local infection with thrombosis of the vessels or as the result of dissemination they may cause more distant effects. A virus may cause muscle damage—for example Coxsackie B.5 causing Bornholm Disease.

(d) pathological conditions such as osteomyelitis, Paget's disease of bone,

cysts, or tumour formation. Trivial injuries often lead to pathological fractures.

In all cases of injury, traumatic effusion results. The form it takes depends upon whether blood vessels are torn at the time of injury, or whether there is simple exudation or transudation through the vessel walls. ([1]Florey, 1958.) The contents of these effusions may lead to the development of fibrous tissue changes if normal reabsorption does not or cannot occur.

Traumatic effusion is derived from:—

1. The excessive accumulation of fluid in the tissue spaces resulting directly from the increase in permeability of intact vessel walls. (Yoffey and Courtice 1956).[2]

2. Ruptured blood vessels. An artery or a vein by a relatively trivial injury may be torn and the amount of fluid in the tissue spaces may be enormous, far exceeding the needs of repair or the ability of the tissues to reabsorb it. It naturally depends on the size of the vessel torn, and on whether it is completely torn. If the tear is complete, the circular and longitudinal muscle fibres in the walls of the vessel can contract and retract to the full extent.

The word 'fluid' has been used because, during the initial examination, the nature of the fluid may be in doubt; but it is, of course, more properly called a haemorrhage, which may be diluted by exudates and transudates from neighbouring vessels or by synovial secretions.

3. In injuries to joints, tendon sheaths or bursae, there may be added synovial secretion derived from increased formation of synovial fluid. This secretion may act as a protection to the articular surfaces; but, in excess, it can make for laxity of the joint, because it has the effect of stretching the capsule and ligaments. If the surrounding muscles should waste, the joint may become even more unstable. A chronic synovitis or capsulitis (with or without adhesions) may eventually develop, especially if the original injury is left untreated.

When structures such as tendons, ligaments, or synovial membrane are torn, they are repaired with fibrous tissue which is formed in response to chemical substances released from the injured tissue. Some observers believe that dead muscle fibres can be re-formed, but the majority opinion is that those which have been destroyed are replaced by fibrous tissue. At first excess fibrous tissue may be formed but later this excess is reabsorbed. In many circumstances, however, fibrous tissue is formed in the surrounding structures, producing adhesions and thickenings which may give rise to pain

and limitation of movement. In some cases it is formed in parts where there is no muscle action and in areas which are called 'dead areas'; and here the traumatic effusion, if still left unabsorbed, is liable to form tender, painful thickenings. These 'dead areas' are situated in such places as the outer edge of the erector spinae where the layers of the lumbar fascia converge.

DIRECT TRAUMA

Direct trauma, such as a blow or knock causes contusion or bruising. If the impact is slight, the tissue may be contused or bruised only superficially. If it is heavy, there may be considerable tissue destruction with disintegration of cells and replacement by fibrous tissue. These fibrous tissue scars may become tender and sometimes account for slow recovery. Skin may be so badly contused that it dies and a slough forms; and this has to separate before healing can really begin. Similarly, if deeper structures are damaged, the cells, depleted of their blood supply, may die. The separation and the replacement of this dead tissue is often a lengthy process.

Tissue disintegration, apart from making the tissues tender, also results in oedema formation. This oedema fluid contains enzymes and special scavenger cells which remove the dead tissues and allow the area to begin to heal.

If a large haematoma forms, part of it can be expressed by operation or aspiration, and in many cases the residue is absorbed. It is not uncommon, however, to find that the residue may cause a tissue reaction, with outpouring of serum which may have to be repeatedly aspirated, or a cystic swelling may form.

INDIRECT TRAUMA

Indirect injury may result from the stretching or tearing of a muscle, or the sudden twisting or sprain of a joint beyond its normal range of movement. First the muscles take the strain, and if the injury is sudden enough to take them off guard, the strain may pass through the muscles to the neighbouring joint, stretching or tearing ligaments and tissues on one side and compressing and contusing tissues on the other.

Muscles are often injured by indirect violence because they are in a state of relaxation or atonia so that they are taken off guard. When they are fatigued or cold, the fibres are vulnerable, because their contraction rate is impaired; and injury may then result from an apparently trivial jerk.

Strong contractions sometime produce severe strain or even rupture of the

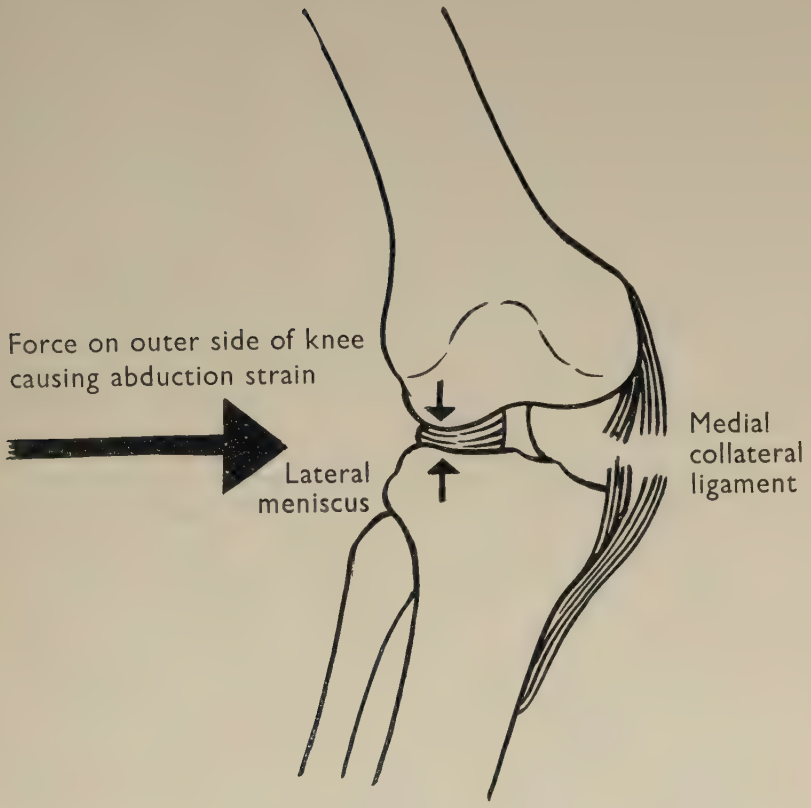

FIG. 1. This illustrates how an abduction strain of the right knee can cause an indirect injury to the medial lateral ligament and a direct injury to the lateral meniscus and lateral side of the joint, by the compressing forces of both lateral condyles.

muscle if they are resisted by a powerful force. If a heavy weight, for example, is unexpectedly thrust into the hand, the long head of the biceps brachii may occasionally rupture: if strong resistance is suddenly withdrawn or, being anticipated, does not materialise, similar damage may occur. For example, when a tennis player makes a backhand stroke and misses, the power developed by the actively contracted forearm muscles and triceps meets no resistance, with the result that they may be strained or torn. The strain may even pass through the muscles and involve the elbow joint.

The 'step that isn't there' is a similar source of injury.

FACTORS WHICH INFLUENCE ATHLETIC INJURY

Postural and occupational strain

The effects of strain resulting from faulty posture have already been stressed (see Part I, Chapter 3). It has been discussed how muscles, particularly the antigravity ones, become tender, possibly because of altered circulation and chemical changes. Gradually, over a period, strain is thrown on the joints which the muscles should be protecting and these too, begin to show symptoms of strain, first in the ligaments and fibrous capsule, secondly in the synovial membrane, thirdly in the articular cartilage and meniscii if present, and finally in the bone itself. These changes may promote periodic attacks of protective spasm, usually in more than one muscle. In time osteoarthritis develops, and at this stage the muscles begin to undergo definite fibrotic changes.

Chronic postural strain produces an accumulation of chemical substances which make the antigravity muscles tender. If the accumulation is sufficient the muscle may eventually be prevented from functioning properly and go into spasm. If a joint becomes strained and one of the intra-articular structures becomes deranged in some way the associated muscles also go into spasm. This spasm is protective to stop the joint being used.

An individual muscle may, therefore, go into protective spasm as a result of:—

(a) accumulation of waste products in the muscle itself
(b) changes in the joint which it should be protecting.

Here, usually more than one muscle is involved.

The ill effects of postural strain may be seen in athletic injuries no less than in the accidents of daily life. As we pointed out in early chapters, the remedy is education in the correct posture which procures amelioration of symptoms and improvement of athletic performance. Good posture aids the circulation while poor posture impedes it. Even though the arterial blood supply may be normal, stasis of venous blood, lymph and tissue fluids may occur if there is poor tone in the antigravity or prime fixing muscles. The symptoms of stasis are swelling, loss of function and tenderness.

Traumatic effusion

In treating recent injury, we aim to keep traumatic effusion on the move in order to help and stimulate reabsorption. Much work has been done in investigating the mechanism by which fluid accumulates in tissue spaces.

Injury to the vessel wall may cause a chemical substance to be released, either directly as a result of trauma or through a nervous reflex (the axon reflex) which causes the vessels walls to become more permeable. (Guyton, 1961.)[3] The normal balance between hydro-static pressure (inside the vessel and in the tissue spaces), the osmotic pressure exerted by the protein fraction of the plasma and the tissue tension is upset and more fluid escapes until a new equilibrium is reached. (Yoffey and Courtice, 1956.)[4] It can thus be seen that if there is any upset in the permeability of the vessel wall, the hydrostatic pressure, the osmotic pressure, the tissue tension, or the lymphatic drainage, the formation of excess fluid can occur. This is what happens when trauma injures an area without disruption of blood vessels.

The path the fluid takes is influenced by the direction in which gravity is acting, the barriers made by fascial planes, the outline of tissue spaces and the

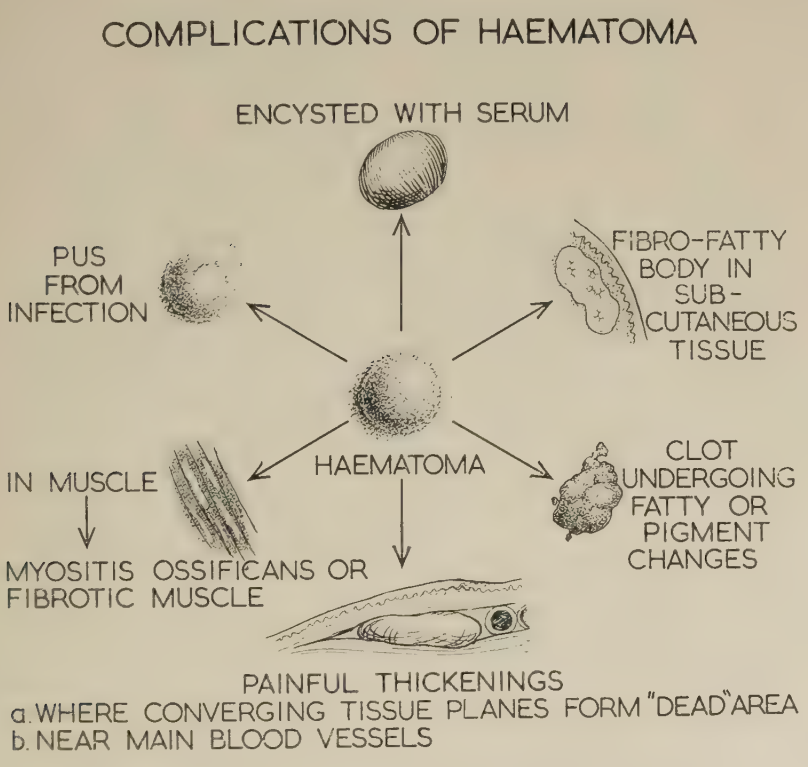

Fig. 2. Complications of haematoma.

massaging action of the muscles, which, of course, influence the direction of lymphatics and venous circulation.

When there is a deep-seated haematoma, active treatment should be begun at once. The treatment ought to be vigorous, but not sufficiently so to cause strain or pain. The products of the haematoma can be traced by palpation as they seep through the substance of the muscle to its surface. Eventually the skin becomes discoloured, or, if there is a large quantity of blood, a fluctuant swelling may appear on the surface of the muscle. It may be possible to aspirate the products through a large bore needle or express the blood clot through a small incision. The aspirated fluid may be blood, serum or serosanguinous fluid. If the haematoma is expressed through an incision, large lumps of clot, like black snails or worms, come out first, followed by tar coloured fluid and small clots.

During its passage through the muscles, some of the effusion may be absorbed. The precise mechanism of the spread is unknown, but it is likely to be a gradual seepage through the tissue spaces and tissue planes, rather than an intercellular exchange. Some of the traumatic effusion may reach the deep layers of the skin before it becomes absorbed; and by this time it may be far removed from the site of the injury. The tissues through which it passes may become tender and swollen and may lose their function, either directly or indirectly as a result of released metabolites. A good example of the seeping and spreading of traumatic effusion is the extensive swelling and bruising that may appear round the ankle in injuries to the calf or even the ham-string muscles.

The lymph and serum components of the traumatic effusion are absorbed quickly, leaving behind a débris of fibrin and cells to be broken down and eventually cleared away by phagocytic action (Florey, 1958).[5] In a joint this débris may become deposited over the joint surfaces as inspissated consolidated particles, which later form as adhesion and tender thickenings, or cause alteration in the articular cartilage.

Any haemorrhage into the tissues must be derived from a broken artery or a vein. If the blood is allowed to stay where it is, it will clot; if it is agitated by movement and physical treatment, the haematoma may break up into small clots which settle in the surrounding muscles. In either case there will be tenderness and swelling which may be connected with a blood vessel and which may therefore simulate a thrombosis. There may even be thrombosis in and around the damaged vessel. The fear of liberating pieces of clot into the circulation may prompt the doctor to prescribe absolute rest. The haematoma is bound to clot, with subsequent fibrosis and disability, and weeks of inactivity for the patient.

Menkin[6] has described many substances which may be released from injured tissues or be formed at the site. There is evidence to suggest that leukotaxine (which he has crystallized) may be responsible for the stimulation of the migration of leukocytes. He has also isolated two other substances which he calls necrosin and pyrexin.

Ageing

The frailties of human tissues show themselves as the result of stresses and strains, the effect of bacterial infections and toxic agencies of various sorts. Ageing is inevitable. The degeneration caused by 'wear and tear' continues from birth to death, but in youth our tissues are so versatile that they can not only repair and replace, but even grow. As people age, however, the wear and tear process gradually overhauls the capacity to repair. The causes of ageing are manifold, but they are mainly degenerative changes in small blood vessels which cause alterations of the blood supply.

Heald[7] and his workers have shown how true fibrositic changes occur at the origin or at the insertion of muscles. They also suggest that, as there is free anastomosis between the veins on the surface of the bone through venous channels to the interior of the bone, changes in these vessels may account for the degenerative bone changes of osteoarthritis.

Furlong,[8] in an endeavour to investigate the pathology of tennis elbow, has excised and examined histologically pieces of tissue from the tender area near the lateral humeral epicondyle. He describes the tissue, which represents the origin from the bone of the extensor muscles, as a mixture of muscle and tendon. He states: 'the tendon is degenerate. The cells stain poorly. There are hyaline areas, occasional small haemorrhage and patches of fibrosis.' The picture is one of local degeneration with gradual replacement by scar. This then is the lesion of tennis elbow—degeneration of musculo-tendinous origin. The cause is unknown.

This is a most important finding, and similar conditions with the same pathology may occur in other parts of the body. It may be that postural and occupational strains interfere with the local circulation of the muscles, and that Furlong's microscopic changes take place in them. Degenerative processes occur in tender structures such as the supraspinatus in tendonitis; possibly they are the cause of tender myalgic spots at the junction of the intercostal muscles with the ribs and sternum, and perhaps of stenosing tenovaginitis. In some patients, however, there may be an actual thrombosis of a small end artery, as the result of vascular spasm or the presence of an embolus.

Fibrosis of the tissues is, unfortunately, a frequent sequel of injury or degeneration. Before healing is complete the process of repair passes clinically through three stages: the acute, the sub-acute, and the chronic. It is considered now that the good effect of hydrocortisone injections is due to their anti-inflammatory properties. The increased permeability of the blood vessels accelerates tissue healing by rapid absorption of the products of inflammation.

Traumatic effusions promote tissue reaction, and in muscles and tendons this may account for:—

(a) weak and tender scars

(b) the formation of muscle swellings of one of the following sorts:—a muscle hernia due to a bulge of the muscle through its sheath; an encysted haematoma in the substance of the muscle; swelling and retraction of the muscle fibres following a tear; hypertrophy of part of the muscle above the tear (see Fig. 1, p. 323, and Figs. 2 and 3, p. 324).

(c) adhesion of the muscle to surrounding structures.

(d) calcification in the muscles or tendons.

(e) tender thickenings on the surface of the muscle.

(f) mucoid degeneration of the muscle or tendon.

In joints, the result of fibrosis may be the formation of adhesions and tender thickenings in the capsule and synovia which cause joint stiffness and limitation of movements. Other structures, such as the skin, nerves, arteries and fascial planes come under the influence of true traumatic fibrositic changes. Fibrosis of muscles and tendons also occurs as the result of tissue degeneration and, if extra strain is thrown on them when they are in this state, they will tear or strain more easily.

Fibrous tissue

The fibrous tissue which is laid down as the result of injury consists essentially of the same protein—collagen—which forms normal connective tissue: but whereas in normal tissue the collagen fibres are orientated so that they provide the greatest mechanical efficiency, in a post-traumatic scar the arrangement of fibres is conditioned by factors which have nothing to do with function. It is only later that the fibres are laid down to cope with the various stresses and strains. It is not surprising, therefore, that fibrous nodules formed after injury should interfere with function and give rise to incapacity and pain.

The main object in giving physical treatment to these nodules is either to disperse them or to impose a functional orientation upon their constituent fibres. A rational basis for therapy is provided by recent work on the chemical

nature of collagen. Collagen fibres are made up of very long molecules composed of fully extended chains of carbon and nitrogen atoms (Lloyd, 1941.)[9] The chains are combined linkage—salt links and hydrogen bonds—which are effective, not on account of their intrinsic strength, but because of the large numbers the parallel arrangement of the main chain renders possible. In the presence of water, the linkages are readily susceptible to ionic penetration and consequent disruption. Collagen does, in fact, owe its name to the ease with which it may be changed from a structured protein into an amorphous gel by mild hydrolysis, a process by which the long main chains became torn away from one another. The physiotherapist must attempt to do much the same thing by milder methods.

In the first place, the aim should be to increase the fluid content of the lesion; in other words, to produce some degree of local oedema (Day, 1948).[10] The oedema fluid should be of an ionic composition which will favour the separation of each of the long chains of the collagen molecule. It is of some interest that physiological concentrations of sodium chloride are sufficient to produce considerable hydration of fascial connective tissue and marked separation of the collagen fibres. There can be little doubt that many of the accepted techniques of physical treatment owe their efficacy to the production of localized oedema and its physico-chemical consequences. When the

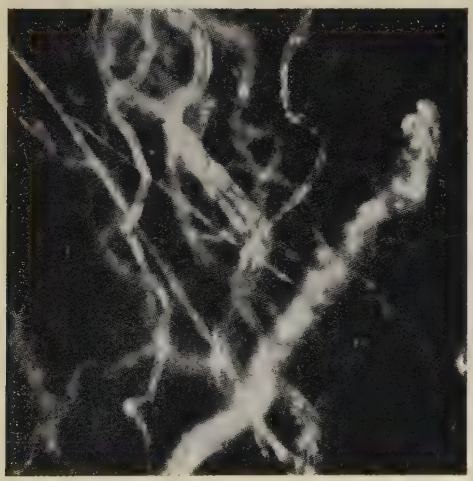

FIG. 3. Rat connective tissue in distilled water at *p*H 7.0: middle of the preparation. It consists mainly of fibrils less than 1μ thick, partly free and partly combined in compact bundles 2-10μ thick. x500. (T. D. Day; *The Journ. of Path. and Bact.*, Vol. LIX, No. 4, 1947).

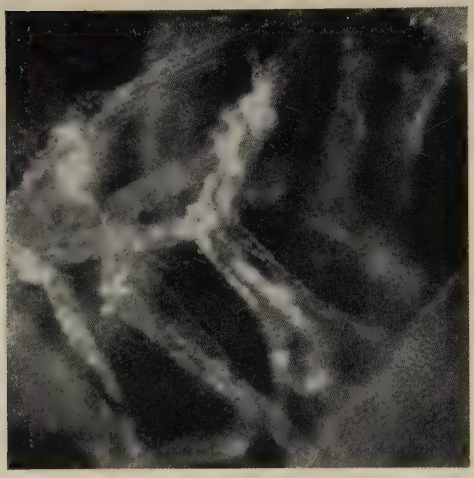

FIG. 4. Rat connective tissue at *p*H 3.6. Two processes are in evidence: Condensation of cementing substance with increased opacity, and acid swelling of the collagen with reduction in opacity. x500. (T. D. Day; *The Journ. of Path. and Bact.*, Vol. LIX, No. 4, 1947).

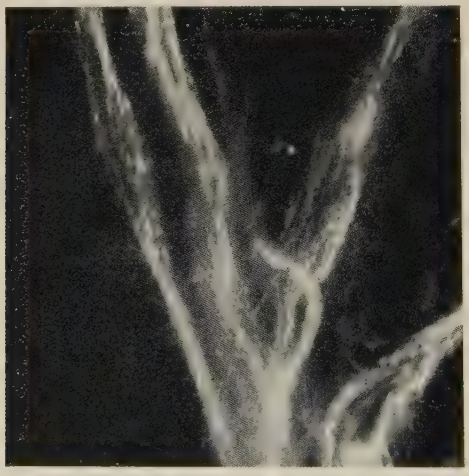

FIG. 5. Rat connective tissue in *M*12 concentration of NaCl. The fibrils in the bundles have become unravelled owing to thinning of the cementing substance. As the result of the reduction in viscosity of the cementing substance, fibrils in such concentrations of salt frequently show Brownian movement. x500. (T. D. Day; *The Journ. of Path. and Bact.*, Vol. LIX, No. 4, 1947).

lesion has been changed by appropriate treatment from a tight knot of disorientated collagen into a softened, sometimes greatly swollen, plastic mass, it is in a condition in which it may be remoulded. The swelling diminishes so that it no longer interferes with function.

In brief, physical treatment should set up a reactionary exudation containing saline substances which cause hydration of the side chains of the collagen molecules.

The Effects of Heating and Cooling of the Tissues

Heating and cooling of the tissues can cause tissue damage. The various types and degrees of burns caused by exposure to sun are well-known, and extreme cold may end in frost bite.

Exercise on a warm or hot day is less likely to cause injury because the circulation of the body is good. Some athletes with chronic conditions feel worse in hot weather. There are many chronic diseases of which the reverse is true and the patient feels much better in warm weather. One explanation of this aggravation in warm weather could be that the increased circulation softens the thickenings and swellings, and the patient is more conscious of them. It is equally possible, however, that there is increased production of metabolites released as a direct result of increased metabolism during increased temperature. Some of these metabolites have the ability to cause pain. If at this stage the patient were to receive regular physiotherapy and carry out home treatment conscientiously there is normally a rapid improvement.

On cold days the circulation is liable to be sluggish, particularly in muscles and joints which have previously suffered strain or injury. Exercise on such days may produce a recurrence of trouble.

Draughts: What exactly happens when an individual sits in a draught and later develops low back pain is not accurately known. One explanation is that the surface blood vessels constrict, and this produces a reflex dilation of the deep vessels, with exudation of plasma and serum. The exudation causes an inflammation of vertebral joints with subsequent protective spasm of the lumbar muscles. In these cases, during phsyical treatment, especially massage, it is easy to observe that there is a substance in the muscles which has made them tender. This substance seeps and spreads through the tissues in exactly the same way as traumatic effusion.

Tender areas

The possible causes of tender areas have been discussed and now the different types of situation in which they appear are summarized:—

(a) Those which appear in relation to the antigravity, static or prime fixing muscles. The area of tenderness may be spread diffusely throughout the muscle at first, but, in time, special areas appear at the side of the muscle where the fascial layers converge, or at the origin and insertion of the muscle. Each of these areas of tenderness may appear during the natural process of healing.

(b) Those which appear in relation to the origin or insertion of muscles associated with recreational disease such as tennis elbow. These may be due to chemical or primary vascular changes.

(c) Those which appear in so-called 'dead areas', due to organization of the products of traumatic effusion. Small nerve endings can grow into these tender thickenings which sometimes become exquisitely tender.

(d) The products of traumatic effusion, as they seep through the tissues, cause tender areas, especially if they are in contact with the periosteum.

(e) A muscle in prolonged spasm can become tender. This may be due to alteration of its blood supply and chemistry. This occurs in postural conditions.

(f) There are tender points which appear at the costosternal junction. They may be the result of postural and occupational strain of the intercostal muscles. Degenerative vascular changes may be present in the areas and there may be an associated arthritis in the neighbouring joints. When these points are treated by massage, one can feel granular, gritty material, which gradually disappears as it spreads. The seepage or spread is exactly similar to that which occurs in traumatic effusions. Other physical methods, such as various forms of heat, anodal galvanism, faradism and shortwave, seem to make them disappear by alteration of their blood supply.

(g) Tender areas may form as the result of exudative fluids which appear in the tissues after exposure to cold or draughts.

SUMMARY

1. Direct injuries can cause tissue damage. There are various degrees from mild contusion and bruising to death of the tissue. Direct injury can break blood vessels and cause large extravasation of blood or plasma.

2. Indirect injuries cause the tearing of the tissue on one side, and compression and contusion of the tissues on the opposite side. Blood vessels may be torn and the resultant haemorrhage add to the bulk of an effusion.

3. Postural and occupational strains have a pathology of their own and are often a pre-disposing cause of acute injuries from relatively minor trauma.

4. Interference with the circulation in any way may be a contributory cause of injury.

5. Tissue alterations of a histochemical nature occur before morphological change.

REFERENCES

1. Florey H. 1958. *General Pathology*. Lloyd-Luke (Medical Books). London.
2. Yoffey and Courtice, 1956. *Lymphatics, lymph and lymphoid tissue*. Cambridge, Harvard University Press. 2nd edition, p. 90.
3. Guyton, A. C. 1961. *Text book of medical physiology*. W. B. Saunders Co., U.S.A.
4. Yoffey and Courtice. 1956. *Lymphatics, lymph and lymphoid tissue*. Cambridge, Harvard University Press, p. 65.
5. Florey, H. 1958. *General Pathology*. Lloyd-Luke (Medical Books), London, Chapter 4.
6. Menkin, V. 1937. Mechanism of Inflammation. *Arch. Path.* 24, 65.
7. Heald, C. B. 1951. The Treatment of Fibrositis. *The Lancet*, Oct. 13, p.659.
8. Furlong, R. *Practitioner*, Sept. 1953, p. 320.
9. Lloyd, D. J. 1941. *J. Soc. Dy. Col.*, Bradford, 57, 281.
10. Day, T. D. (1947). *The Journal of Pathology & Bacteriology*, Vol. LIX, No. 4, pp. 56–573.

3

TREATMENT

Some general principles

THE first principle of treatment is to start early. In the middle of the last century Hilton[1] produced his book 'Rest and Pain', and it made such a profound impression on the profession that, even to-day, the distinction between complete rest and periods of rest followed by gentle graduated exercise is not always made. There is an ideal balance between the amount of rest and movement required in each case. In the early stages following severe injury, long periods of rest followed by short periods of activity are indicated; but as recovery takes place, longer periods of activity can gradually be undertaken until full function is restored. Dispersal of traumatic swelling is relatively easy in the early stage because it is mainly fluid. If treatment is postponed even for a few days, the swelling may start pathological processes other than those of healing. Much valuable time is lost, to the discomfort and inconvenience of the patient.

Hilton's concepts were intended to apply to structures inflamed as a result of infection. He rightly stressed that the barrier put up by the patient's natural defences would be broken down by movement, and the infection would be likely to spread locally or even become a septicaemia.

Gradually, over the last century, surgeons, Tucker (1935)[2], Perkins (1952)[3], have come to appreciate that inflammatory conditions due to injury should be treated entirely differently from those due to bacterial infection.

In the early stages of injury, even if the synovial membrane of a joint has been involved, movement can be started so long as it is without strain—in the leg, for example, without full weight-bearing. As the condition improves, strain can be gradually introduced: in fact, provided it is not excessive, this strain helps to promote normal joint circulation. Trueta (1954)[4] has shown that the articular cartilage of weight-bearing joints is healthiest when it is compressed regularly, as in frequent weight bearing. Movement and compression are necessary for the uninterrupted circulation of the synovial fluid.

Isometric and isotonic muscular contractions

An important distinction in active movement is the difference between

isometric and isotonic contraction of the muscles. When an injured limb is in plaster of Paris, contraction of the muscles against the resistance of the plaster is isometric; that is to say, the muscle fibres contract and broaden but do not shorten. If the joint is in the optimum position of rest, movements of this sort prevent wasting of muscles and over-stretching of the associated anti-gravity muscles. They also promote the absorption of traumatic effusions.

In isotonic contractions, on the other hand, the tone in individual muscle fibres is equal, but their length changes.

An adequate wrist-joint or ankle-joint plaster should allow both types of movement—the muscles acting on the wrist or ankle joints contract isometrically, but the fingers and toes move isotonically.

When a fracture involves the articular surface of a joint, the injured joint is placed in a plaster of Paris cast which enables the overlying muscles acting on that joint to be contracted isometrically: but the contractions cannot be entirely isometric, for so tight a plaster would damage the circulation. The isometric contraction of the muscles is, in effect, the static contraction of the muscle fibres which fix and support the joint, as in 'Active Alerted Posture' (see Chapter 3). When the plaster is removed, active exercises are carried out up to the limit of painless movement. Strain of any sort must be avoided.

Treatment clinics

In the last twenty years, many large sporting organizations have established medical units of their own. The professional football clubs, for example, are well equipped to deal properly and expeditiously with injuries: but the great majority of sportsmen have to take their own initiative in securing treatment. However minor the injury, athletes should be encouraged to seek expert advice at once, so that they can be restored to fitness as soon as possible. On the other hand, when an injury occurs on a Saturday afternoon, it is not always possible to get such advice until after the weekend. The athlete, himself, therefore should become familiar with simple methods of treatment, which do no harm but can help him to hasten recovery; or at least to give him relief until he is able to obtain medical attention.

Doctors who treat athletic injuries must keep it in mind that most athletes are anxious to return to their sport as soon as possible. Some injuries, however well they are treated, take a considerable time to improve and it may be several weeks, or even months, before the patient can return to active sport. It is, therefore, essential to enlist the athlete's whole-hearted co-operation by explaining carefully to him the exact nature of his injury, the form which treatment should take, the earliest possible time in which recovery can be

expected, and the complications that could arise unless they are forestalled by appropriate treatment. As a rule athletes have a most sensible approach to their injuries and do all they can to get well. Disabilities due to functional causes or malingering are extremely rare: the commonest difficulty is to curb their keenness to return to play.

Recovery depends on many factors—the type and extent of the injury, the nature of the game, the particular occasion for which recovery has to be made, and the age of the athlete.

A cricketer with an injured hamstring muscle is incapacitated longer if he is a fast bowler than if he is a batsman or wicket-keeper. Similarly a hand injury is liable to keep a goalkeeper idle longer than a centre forward.

For some games, particularly international events, the athlete must be one hundred per cent fit: he cannot risk letting his side down and impairing his reputation by returning before he is fully recovered. Selectors, especially of international teams, ought to make it a rule that, after an injury, a player is chosen on current form. It is common experience that the limitation of activity during recovery from an injury causes some loss of form, so that the player is temporarily not up to international standard.

The Effects of Age

In athletics a man reaches his prime at about twenty-five; in lawn tennis a year or so later; in football at about twenty-eight; in cricket and golf at or after thirty. During the third decade, wear and tear begin to show their effects, and by fifty there is clear evidence of deterioration. In certain events, such as long distance running, one may postpone the deterioration of performance if there has been regular and continuous practice; and whatever the sport, training must take account not only of the degree of efficiency needed, or the period of time the effort has to be maintained, but also a man's age. But as he gets older, even the athlete who has kept himself in training must realize that his tissues are not as resilient and supple as they were when he was twenty. Through the normal process of ageing, the muscles, ligaments and areolar tissues gradually lose their elasticity and, therefore, are more liable to be torn or overstretched; and, once they are injured, however well they may heal, they are never quite normal again. For ordinary life and work, 75-80 per cent recovery of muscles and joints is ample to avoid aches and pains or disability: but moderate exercise like tennis, cricket, golf, or squash, demands 85 per cent to 90 per cent recovery; and international events in any competitive sport, demand the full 100 per cent.

Musculo-skeletal efficiency diminishes about 1 per cent per year after the age of twenty-five; so that the athlete at fifty is only 75 per cent as effective as

he was at twenty-five—that is to say, he can achieve by maximum effort at fifty the level of performance which was his normal everyday level at twenty-five. If he attempts more he strains or hurts himself. At sixty, though he may be 100 per cent fit for his years, he has lost a further 10 per cent efficiency; so that his maximum now cannot even reach his 'ordinary' level at twenty-five. These figures of course are both arbitrary and approximate, taking no account of individual variation. Borotra, for example, at sixty can still beat opponents years younger: but his form is nevertheless about 30 per cent below what it was when he was Wimbledon champion.

The older one gets, the longer the interval between exercise and the appearance of muscle and joint stiffness. At twenty, it often appears within a few hours; at twenty-five the next day; but at fifty it may not appear for two or three days, perhaps because in youth the changes are in the muscles, but as one gets older the changes are deeper, in the joints. But at all ages there is a latent interval between the exercise and onset of muscle stiffness and pain. The scientific reason for this latent interval has never been adequately explained and is ill understood.

Negative and Positive Phases in Healing and Recovery

Injuries whether caused by direct or indirect violence may, by cutting off the blood supply or by actual contusion and bruising of the tissues, result in death of certain cells. In all cases there is traumatic swelling, and although some of it goes towards natural repair, excess is harmful. Repair is chiefly by organization of some of the products of traumatic effusion which results in the formation of fibrous tissue.

Two phases can be discerned during treatment, the negative and the positive. The use of the term 'negative' implies that there are present in the tissues traumatic swelling, tissue tearing and even tissue destruction. The original condition may even deteriorate and the symptoms worsen before signs of recovery appear. This phase occurs where there has been extensive contusion and bruising of the structure so that dead tissue has to separate before healing can take place. In trauma, this separation and elimination of damaged structures form the negative phase, and as soon as it is complete, the process of healing can begin, that is to say, the positive phase sets in. The best way to shorten the negative phase may be surgical removal of dead tissues and traumatic swelling.

Alternatively, an injury can be described as passing through the following stages:—the acute, the subacute and the chronic. If active treatment is given correctly from the outset, the injury should not be allowed to progress to the chronic stage, which means the tissues have become fibrotic with adhesion

formation and there will also be loss of function of the injured part. The whole object of active treatment is to prevent the chronic stage. The negative (or catabolic) phase coincides with the acute stage and the positive or anabolic phase coincides with the subacute stage through to the restoration of the tissue to normal.

THERAPEUTIC FUNDAMENTALS

The therapeutic object in the treatment of injuries should be the restoration of the damaged tissue to a condition as nearly normal, both structurally and functionally, as before the accident. The basic methods which ensure good healing are:—

1. Support of the injured part, which must be rested from all forms of strain but, from the beginning, exercised gently without strain. This may mean avoiding lifting objects with the upper extremities and weight bearing in the lower extremities.

2. Methods directed to improving the circulation in order to bring to the injured part a healthy blood supply. This assists re-absorption of traumatic swelling and at the same time, facilitates repair of the tissues.

3. The control of the amount of traumatic swelling.

4. The removal of the products of tissue contusion; and the dispersal and absorption of the excess of traumatic swelling, so that fibrous tissue reaction is restricted to just that amount which is necessary to heal the damaged structures.

5. The prevention of the formation of adhesions and thickenings and the production of a supple scar, resilient and mobile on all surrounding structures.

6. The establishment of normal muscle tone and power with full extensibility and flexibility of the individual muscle fibres. If possible, it is important to restore the muscles to a stronger condition than before the injury, so as to help prevent recurrence of the injury under similar strain.

7. Restoration of co-ordination of movements by graduated training. Progressive resistant exercises are useful in this respect in the later stages of training.

In many instances these basic principles are directly dependent one on the other.

PLANNING TREATMENT

The doctor is well advised to have a comprehensive plan which can be applied to all injuries. It should cover:—

1. preventive treatment
2. first aid treatment
3. treatment by doctor or specialist
4. treatment by physiotherapist
5. home treatment by the patient
6. rehabilitation. If the patient is an athlete, the programme can be supervised by his trainer; if he is an industrial worker, by his welfare officer, and if he has to stay in hospital, by the occupational therapist.

It must be impressed upon the patient and his attendants that, at each stage, the most minute attention to detail is essential, and that only if the instructions given are executed precisely can good results be anticipated.

The surgeon must be prepared to see his patient almost daily, and he must evolve a programme of treatment which gives the patient confidence that everything possible is being done to hasten recovery. Conservative measures are usually indicated but sometimes an operation is essential. Whatever the scheme of treatment, it must be adapted to the individual needs of each patient, who may have peculiar problems or requirements which he should be asked to explain at the outset. It is then up to the surgeon to decide when the patient is fit to take up his chosen activities. A cricketer, for example, with a torn hamstring muscle, may be able to start batting in a few days afterwards, provided treatment is started at once, and the muscle is firmly supported; but if he is a fast bowler, the torn muscle must be healed completely and subjected to graduated stress and strain before he can be passed fit: and this may take four weeks.

Another important factor which should be considered is the time at which the injury occurs. If an athlete breaks down two or three weeks before the end of the season, it may be possible to patch him up sufficiently to carry on, if that is what he wants. If he fails, he has the whole of the closed season in which to recover. On the other hand, if an athlete breaks down at the beginning of the season, he ought to be advised to wait until recovery is complete, even if it means an operation and four to six weeks lost out of the season.

PREVENTIVE TREATMENT

Cure is all very well, but how much pain, disability and wasted time we can spare the patient if we can prevent injury!

Some of the most important preventive measures are as follows:—

(a) The athlete should have a good knowledge of correct body posture and body mechanics. This is best inculcated at an early stage, preferably by the age of twelve, when most young people are ambitious to become great sportsmen.

(b) There should be a thorough and efficient training programme before any major event, which should include a regime of correct diet, plenty of rest and sleep. Practice of the sport alone is often insufficient to ensure protection from the sport's special hazards. Additional basic strength training is also often necessary.

(c) Suitable clothing should be worn for training and the actual event. The player should keep warm beforehand and go on to the field with the circulation so stimulated that all parts of his body receive an adequate blood supply the moment they are exercised.

FIRST AID TREATMENT

In every athletic event there should be an official charged with the responsibility of deciding whether a player who has received an injury is in a fit state to continue. Unless he is taking part in some vital event such as a cup tie, the player should stop at once or further damage is almost certain. If the damage is severe enough he will have to stop anyway.

The essentials of first aid are as follows:—

(a) treat the general condition, such as shock or concussion:

(b) protect and support the injured part so that the condition is not aggravated:

(c) control traumatic swelling by firm bandaging and the correct positioning of the injured part:

(d) get the injured man to the doctor as soon as possible.

It may not be possible always to observe these principles, but they are excellent as a general guide to action. It is often necessary to improvise, using what is available at the time for protection, support and splintage.

TREATMENT BY THE DOCTOR AND SPECIALIST

When a diagnosis has been established by clinical examination and such other ancillary means, as are necessary, treatment should start at once, along whichever of the following lines apply to the particular case:—

General treatment

General treatment should include the treatment of shock and the reduction

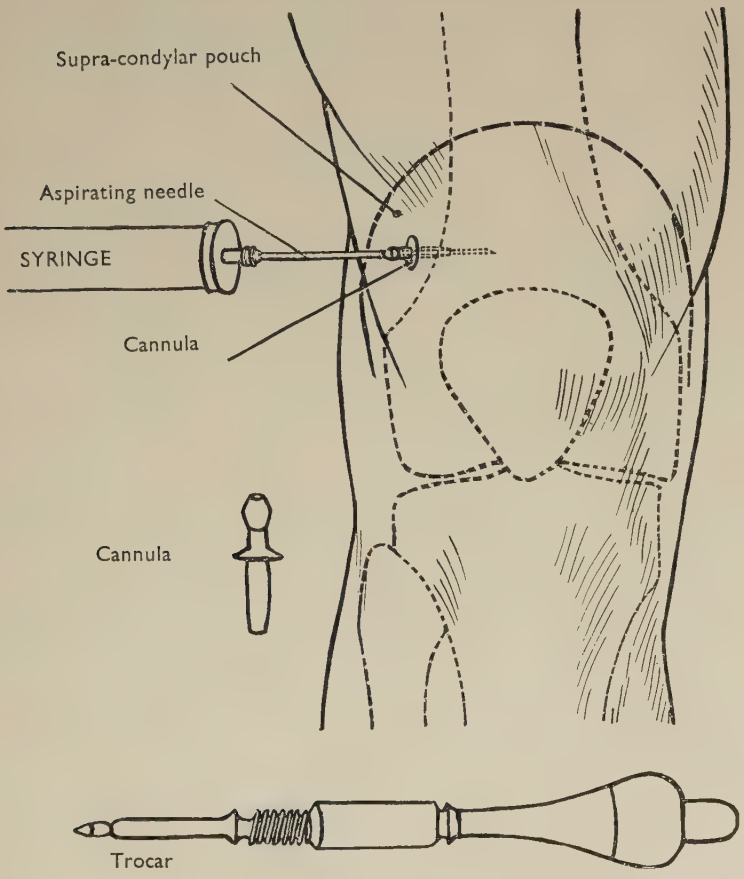

FIG. 1. Aspiration of synovial fluid or blood from a knee joint. Local anaesthesia (5 ml. 1% xylocaine) is first injected in point on the outer side of the joint just above the patella; a small nick is made to insert the trocar and cannula, and through the cannula a suitable needle is inserted into the suprapatellar pouch.

of pain by analgesics. Metabolic conditions such as gout, septic conditions and toxic foci should be excluded.

Local treatment

(a) the correct positioning of the limb:

(b) the protection and support of the injured part by a suitable bandage and splinting:

(c) the aspiration or expression of excess traumatic swelling:

(d) the injection of procaine or xylocaine with hydrocortisone, or urea and salicylic acid solution: a small amount of hyaluronidase may be added to any of these solutions:

(e) the gentle manipulation of the injured part after the muscles have been releaxed by heat, ethyl chloride spray or by local injections.

(f) detailed and precise instructions to the patient as to the amount and degree of rest and movement: the object is to rest the damaged part from strain, at the same time, to encourage gentle muscle activity without strain: later one can add light strain which is increased as recovery proceeds: he should also be given definite instructions about the other forms of home treatment:

(g) operation without delay, when it is indicated:

(h) detailed instructions to the physiotherapist.

TREATMENT BY THE PHYSIOTHERAPIST

Various forms of physical treatment are available and usually are given in combination. They include:—

Heat—by lamp or shortwave diathermy
Massage
Faradic stimulation
Manipulative therapy
Anodal galvanism—sinusoidal
Ultrasonic therapy
Deep X-ray
Interferential therapy
Proprioceptive neuromuscular facilitation.

The physiotherapy department, however, must not be considered the cesspool for chronic cases, but a very active department. The physiotherapist must make certain that the patient is performing his exercises correctly and

walking properly. The doctor must always give the physiotherapist precise instructions upon the type and duration of treatment. The physiotherapist will note changes from day to day in the physical signs. These may coincide with changes in symptoms, and his observations enable the doctor to adjust the treatment according to progress.

HOME TREATMENT BY THE PATIENT

This should include:—

Rest from weight-bearing or strain—complete, minimal, or moderate
Exercises without strain
Home massage
Poultices
Support
Medicinal therapy

Apart from organized physiotherapy, the patient must himself carry out home treatment conscientiously and regularly at least twice a day. There is an aphorism which advises the patient to carry out his home treatment 'with vim and vigour, religiously, regularly, resolutely, if somewhat reluctantly.' A convenient way to give him the instructions he needs is a standard leaflet, written in simple lay terms. He should be encouraged to massage the injured part either by effleurage, kneading or deep friction according to his condition. This massage is combined with contrast baths and hot baths. In certain cases poultices, protection and support should be applied after the treatment.

The patient should carry out remedial exercises of varying intensity and at regular intervals throughout the day. He must assist his own recovery and be made to realize from the first that only by the fullest co-operation with his medical advisers will recovery be effected in the shortest time. Home treatment is considered more fully later in the chapter.

REHABILITATION BY THE TRAINER

In professional football clubs and larger amateur clubs there is usually a trainer. There should be full co-operation between trainer, doctor and physiotherapist. This co-operation should start as soon as possible, so that the athlete may carry on his training as far as he can while the injured part is being treated. This applies particularly to injuries of the upper extremities where a fracture of one of the bones or a severe sprain of a joint will allow the athlete to continue training the rest of his body either running, jumping

or, if he is a footballer, practising ball control. This can take place even if the injured limb is in a plaster of Paris cast.

INDICATION OF PROGRESS

It is sometimes difficult to decide whether to allow the patient to resume normal activity. In such cases, provocative, stimulating treatment can be given as a test. This is called 'purposeful aggravation'. Treatment and exercise of a provocative nature are given, gently at first so that the reaction is a controlled one. If, in joint injury, there is a return of synovial effusion and the patient is kept awake at night, the provocative treatment has been too violent or started too soon.

Morning stiffness is also a good guide to recovery. As the condition improves, so the period of early morning stiffness diminishes. Any general swelling of the part or pain as the result of provocative treatment or exercise is a sign that a slower programme is necessary; but some slight reaction with swelling may be inevitable and the patient should be warned to expect it. It is the natural reaction of the body to form adhesions and thickenings in an endeavour to restore the tissues to normal. If the reaction is marked and persists despite the retardation of the treatment programme, one should suspect a septic focus or some other complication.

MEASURES TO PROMOTE DRAINAGE

Venous, lymphatic and tissue fluid drainage can be materially assisted by the correct positioning of the limb. In leg injuries, the end of the bed may be raised on blocks of about 6 in. or high blocks of 9 in.-12 in. A simple way to get a 3 in. elevation is to put two 1½ in. thick telephone directories under each bed pedestal. Drainage can further be assisted by gentle remedial exercise, which will, by the pumping action of the muscles (an affect sometimes called the 'peripheral venous heart'), aid the returning circulation, and by careful massage. The patient lies reversed on the plinth so that the legs, supported by pillows, are at the raised head end and are considerably higher than the rest of the body. Sweeping effleurage from the toes up the leg may be combined with faradism given with the swollen limb compressed by a pressure bandage. Frequently, by the end of treatment, an enormously swollen ankle and leg become almost normal. Treatment along these lines also assists the chronic effects of vascular disturbances caused by thrombosis or 'white leg'. The arm is easier to drain: it can be slung from the top of the bed or, better still, suspended from a blood transfusion stand.

FIG. 2. Various types of support. (*Clockwise, starting back left*): a split ischial tuberosity bearing plaster; a corselette; shoulder check strap; clavicle rings with abdominal check band attached; tubo foam; felt and elastic webbing bandaging (*front left*).

SUPPORT

It is impossible to overemphasize the importance of supporting a damaged structure. If an injured muscle or joint is allowed to function actively without appropriate support it will almost certainly be further strained. Through injury to a joint the associated muscles are often reflexly inhibited and become atonic; without adequate support these atonic muscles may suffer further strain and the recovery of their tone be delayed.

Suitable, efficient support to the damaged part gives instant relief, helps to prevent recurrence of the injury, allows the muscles to regain their tone and gives the patient the confidence to make gentle movements.

There are varying degrees of support, from the most flexible to the most rigid, according to the type of injury; but even in the grossest injury some degree of movement is beneficial.

The supports advocated below are employed according to the needs of various types of injuries.

1. *Plaster of Paris or plastic support for severe muscle and joint injuries*

A padded plaster of Paris cast is applied and split (but not bivalved) as soon as it is sufficiently dry. This split cast can be easily removed for treatment and replaced afterwards. It is held firmly in position by suitable straps or bandages. A plastic splint has similar advantages.

2. *Orthopaedic felt and elastic webbing support*

This type of support is much used for all types of injuries to the chest and low back, because it can easily be removed for treatment and baths. The pain caused by contusion injuries or fractures of the ribs is greatly lessened by the application of a large layer of orthopaedic felt, over which elastic webbing is firmly, but not too tightly applied. This tends to constrict the movements of the chest but still allows the patient to breathe and use the muscles without undue pain. There is also a proprietary corselet, made in varying sizes, which is much easier to put on and off than the webbing. If obtainable it is to be recommended.

3. *Adhesive plaster or elastoplast strapping*

Adhesive strapping and elastoplast have different uses: these are complementary to one another. Where rigid support is required, strapping is best employed, but it should not be applied to unshaven skin. If the part is not to be shaved, strapping is applied over stockinette or a cotton bandage. Strapping support, combined with pressure pads of felt or sponge rubber, is often employed for joint sprains. Even in this type of injury, a readily removable support is more satisfactory than strapping until the patient is fit to return to his activities. Suitable pads of felt, sponge rubber, or wool, should be applied with firm crepe bandages and, if necessary, over this a figure of eight strapping or elastoplast. The support can be removed for the treatment and physiotherapy which are so vital in the early stage. When the patient is ready to begin exercise, a more rigid support, such as adhesive strapping or elastoplast, may then be indicated.

A torn muscle should be splinted by three or four pieces of waterproof elastoplast applied over its whole length. This may be covered with stockinette and the whole limb encircled with elastoplast, applied firmly but not so tight as to constrict the circulation.

Small joints can be held and supported by waterproof elastoplast or Tubofoam which will not interfere with home treatment such as contrast baths, hot baths and massage, nor with massage and faradism in the physiotherapy department.

None of these forms of support should be left on so long that the muscles become atonic. This applies, particularly, to support in low back conditions. In the acute stage, the patient may need absolute rest, lying passive and well supported in bed. As he improves, he starts graduated exercises still on his back. In the next stage he is allowed to walk in a plaster jacket, which should be a full one coming to within 2 in. of the top of the sternum in front. Some patients recover so quickly that a plaster jacket is unnecessary: a brace such

as the McKee brace, may be adequate. This can be removed for baths, exercise and physiotherapy. After a time in the brace, a low back belt of some kind may be sufficient. As the muscles strengthen through each phase, the more rigid type of support can be discarded for gradually increasing periods until, eventually, the patient requires no support at all. This state of affairs, however, is only reached by training the muscles so that they actively protect and support the joints, through the fixation of the prime fixor muscles.

It has been stressed that no support should be continued if it tends to make the patient lazy or in any way interfere with the normal action of the muscles and joints. The right type of support is one which allows the muscles to appreciate the pressure of the support and react against it. In this way proprioceptive impulses are increased and there is developed a balanced static isometric contraction of the muscles surrounding the joint. Thus the power of these muscles which are the prime fixing ones, is restored, so that once more the injured joints are adequately supported and movements controlled. This is the basis of Active Alerted Posture.

MASSAGE

Platt[4] criticizes the present day tendency to neglect the use of massage. Many surgeons prefer to rely almost entirely upon exercises: but there are conditions in which massage must be combined with exercise and other forms of physiotherapy in order to obtain the quickest possible recovery.

There are various types of massage, such as effleurage, friction, kneading and tapôtement.

The objects of massage are:—

(a) to promote venous, lymphatic and tissue fluid return:

(b) to diffuse traumatic effusion and so assist absorption:

(c) to break down the thickenings caused by the consolidation of the lymph and blood in the tissues:

(d) to keep supple the mobile soft tissues which tend to become stiff and stuck together by traumatic effusion.

In the early stages after trauma, even the gentlest of active movements may be accompanied by pain and spasm; and, as this is the period of maximum swelling, massage assisted by posture, can prove of great benefit. Because of the extreme tenderness of the tissues, effleurage (stroking towards the heart) may be as much as the patient can tolerate. If the massage is performed under infra-red or radiant heat, the pressure and strength of the stroke may be gradually increased; so that within a few minutes the part can be massaged

quite vigorously. Thus products of bruised tissues are encouraged to seep through the tissues and spread from the injured point like the ripple of a wave. Painful spots are noted and the massage is carried out concentrically, the physiotherapist working from the periphery inwards and then outwards.

Where there are chronic, tender thickenings at the origin or insertion of muscles and tendons, deep friction massage across the fibres helps to disintegrate any abnormal thickenings present and to reorientate the arrangements of collagen fibres. This form of treatment is particularly advocated by Cyriax.[5]

MANIPULATIVE SURGERY

Manipulation is a form of therapeutic movement. It should play a most important part in the treatment of all injuries.

Every joint has a small range of involuntary movement, that is, movement which cannot be performed by the patients themselves. This movement allows sliding, gliding and rotation of the bones on each other, and must be complete to allow full voluntary movement.

FIG. 3 Manipulation of wrist. Traction and counter-traction are applied so that the joint surfaces of the proximal row of carpal bones are pulled away from those of the radius and ulna.

To James Mennell[6] the profession owes a great debt of gratitude for putting manipulative surgery on a sound basis. He postulated that the principles of manipulation must be:—Firstly, to distract, that is to pull apart the joint surfaces by traction and counter-traction. This helps to restore any loss of resilience or elasticity of the capsular structures, tendons and muscles and other soft tissues. One hand grips the structures above the joint to be manipulated (the counter-traction), the other hand grasping the structures below applies the traction. One must take great care not to apply too great a leverage.

FIG. 4. Manipulation of wrist. Side bending to the ulna side with traction, counter-traction and pressure of the thumb on the ulna collateral ligament. This manoeuvre opens the space between the radius and scapoid on the outer side and stretches the radial collateral ligament.

Secondly, to carry out the involuntary movements while the distraction is maintained. In the wrist joint, for instance, the ulna and radius are firmly held while the first row of carpal bones with the rest of the wrist and hand are moved sideways, first to the thumb and then to the little finger side. These manoeuvres are followed by movements forwards and backwards. Finally the carpal bones are twisted, in both directions, on the radius and ulna. Not until the full range of involuntary movement is restored is it possible for the

FIG. 5. Manipulation of wrist. Dorsiflexing hand. Traction and counter-traction are maintained. The hand and carpus are dorsiflexed round the lower end of radius and ulna. This is helped by the thumb of manipulator's left hand pushing radius and ulna forwards.

patient to obtain complete voluntary movement. Thirdly, to carry out the voluntary movements, while the distraction is maintained; the distal bone or group of bones is carried forward on the proximal group in flexion and backwards in extension.

These three principles are followed whether the patient is being manipulated gently without an anaesthetic or more forcibly under an anaesthetic.

The manipulator must, through experience, learn never to over-manipulate less he should create too great a reaction which will cause pain and stiffness. If the manipulation is performed without general anaesthesia, the patient may feel a slight reaction and increasing pain on movement for a day or two. If he is warned he will expect the reaction and not be unduly concerned. A marked reaction is only likely if the manipulation has been too violent, or too early (when the condition is still in the acute or negative stage); or if unsuspected sepsis is present.

When a manipulation is carried out under an anaesthetic, it should be performed as a controlled manipulation, that is to say, great care should be taken

FIG. 6. Manipulation of wrist. Plantar flexing the wrist. The carpus with hand is pushed forwards by the thumb around the lower end of the radius and ulna while these bones are pulled backwards. At the same time traction and counter-traction is applied.

to avoid using a long lever, for it is possible to break the bones. If there are tough adhesions, they may fail to give way and the bone may crack instead. This is particularly liable to happen to the humerus when a manipulation is being performed for capsulitis for the shoulder joint. Whether a general anaesthetic is used or not, the correct method is first to take up the slack in the muscles, so that the joint or joints can move no further in the direction in which they have lost full freedom. In order to break down adhesions and restore full movement, the surgeon carries out a quick, sharp movement in the direction of the restriction, so that the final manipulative movement is executed with low intensity and high velocity.

Manipulation under general anaesthesia may cause a reaction which will last for a week to ten days. If this reaction is excessive and lasts longer, there is a danger that adhesions may form again.

One cannot overemphasize the importance of gentleness. In those cases where swelling and oedema accompany the restriction of movement, it is sounder to disperse the oedema and diminish the swelling by gentle manipula-

tions and physiotherapy, before one breaks down the adhesions by manipulation under an anaesthetic. It is in this type of case that too early manipulation under an anaesthetic may provoke further oedema and swelling; there results a violent reaction which cannot be dispersed before adhesions reform. The condition, far from being improved, is aggravated.

The products of the reaction to manipulation must be dispersed by appropriate physical treatment. Sometimes a local injection of some type of anaesthetic fluid with hyaluronidase or cortisone is given at the same time. This often helps to disperse and promote the absorption of the swelling and effects a quicker recovery.

A contracted, shortened muscle frequently requires manipulative treatment. If the muscle has an action on two joints, both joints must be put through the full range of movement. The joint above is fixed so that the muscle is placed on full stretch: then the joint below is put through the full range of movement, and vice versa. In addition, the muscle must be fixed with one hand near the origin, and the belly of the muscle moved from side

FIG. 7. Manipulation of a muscle. One end of the muscle is fixed with one hand and the other part of the muscle is stretched from side to side along its course. Then the process is reversed, fixing the other end. Following these manoeuvres the joint or joints on which that muscle acts are put through a full range of movement.

to side with the other hand. The process is repeated with the insertion fixed.

Manipulation is one of the most important forms of physical treatment: but it must never be in any way violent. Gentleness is the essential quality. It is better to restore involuntary movements a little at a time in successive treatments, rather than to use force and risk delaying recovery.

These manipulative methods are not to be confused with passive movement. When joint movement is examined, the range of both active and passive movements must be explored; but 'passive movement' as a form of treatment should never be employed, because it usually implies forcible movement. Gentle manipulative movement is entirely different: it assists the free movement of one bone on another—the osteopathic doctor calls it 'articulating'.

SHORT WAVE DIATHERMY

A form of heat therapy commonly used is short wave diathermy, which induces within the tissues current of such high frequency that no electrical sensation is felt by the patient. Heat is generated not only on the surface but also in the interior of the injured part. Precisely how it produces its effect or why it is useful, is not at all clear, for so many theories about its action have now been conclusively disproved by the use of radio-active isotopes. But it certainly is valuable, though there are certain contra-indications to its use. It induces vaso-dilation and increases blood flow, and so helps to clear up infected areas; but it should never be employed in any condition in which the tissues are atrophic or where the blood supply is reduced. Nor should it be used where there is a fracture or crack, for it definitely inhibits the formation of callus. In osteoporotic conditions it may cause even further decalcification.

Short-wave diathermy is best given after massage and is extremely useful in treating a joint in the early stages of injury. It is useful, also in combination with deep friction massage, local injections and gentle manipulative movements, in the treatment of local tender thickenings. Extensive bruising of a muscle, fibrositic deep seated thickenings and joints stiff with adhesions all benefit from short-wave diathermy; but if treatment is prolonged, it is liable to make a muscle flaccid and atonic.

Interrupted galvanism

When muscles lose their motor nerve supply they quickly waste and become fibrotic, unless the fibres are regularly stimulated by an interrupted

galvanic current. This type of current can be most uncomfortable and unpleasant for the patient who feels a sensation of burning. The discomfort may be relieved by a wet pad of about eight layers of lint which is applied over the muscles to be treated. To control the 'make and break' of the current, the cathode button is dabbed against the pad. The anodal pad is applied firmly to any dependent part of the body. The muscle contracts at the 'break' and the patient can just feel the movement.

If the intrinsic muscles of the hand are to be stimulated, the area is covered with a large pad, so that each muscle can be given the requisite number of contractions separately. Seddon[7] states that all denervated muscles need at least ninety contractions each day to maintain their volume and tone.

Anodal galvanism still has a place in the treatment of painful periosteal contusion and traumatic synovitis. A small current of 3-5 milliamps only is applied by the through and through method for twenty minutes, the anode being placed over the tender area and the cathode on the opposite side of the joint.

In certain cases the use of drugs such as Renatin or iodine can be placed on the anodal pad and driven in to the tissues by the current to produce a counter irritation and this relieves pain.

Surging faradism: some muscle wasting is bound to occur after every injury, and in injuries to joints wasting of the muscles appears relatively early. As the movements of a joint depend on the muscles controlling that joint, it is essential to restore their full power and tone or the joint is liable to give way on movement and become unstable. In this type of case the application of faradic contractions is most beneficial.

Faradic treatment can be harmful if the electrodes are strapped on in fixed positions and the muscles stimulated by a metronome. The same muscle fibres contract each time and tire quickly. After five minutes of this treatment, the tired fibres may be damaged.

The late Morton Smart[8] developed a method of treating injuries by graduated muscular contractions. He taught that the only effective way to carry out faradic contractions is to 'surge' with one hand and apply the active electrode with the other (the 'labile' method).

Every injured muscle has its own rate of contracting and relaxing. If the physiotherapist passes from one muscle to another giving each five to six contractions, there is no chance that he will tire any one muscle. He can observe the characteristics of individual muscles, and allow them to contract and relax at their own rate. Meanwhile the patient sits or lies comfortably with the muscles under treatment completely relaxed.

It is often said that active exercise does more good than faradic stimulation of the muscles. This statement is on the whole correct; but after some injuries, active contraction of the muscles is often so painful at first that the patient cannot carry them out. In such cases gentle graduated faradic stimulation helps to restore normal function. Certain loose, unstable joints, also, can be made stable by daily faradic contractions combined with graduated active exercises, a result seldom achieved by active exercise alone.

Ultrasonic radiation

Ultrasonic waves are those acoustic frequencies between 20 and 500,000 or more kc. The wave length at the extreme upper range approach those of visible light. Unlike electromagnetic vibrations they may be propagated though a vacuum and travel four times as fast in liquids and twenty times as fast through solids as they do in air. These waves travel in straight lines and can be completely reflected at the interface of two mediums.

Ultrasonic vibrations induce mechanical chemical and thermal effects. The destructive action of ultrasonics is due to the formation of gas bubbles by cavitation.

The biological effects have been extensively investigated. Hyperaemia, oedema, vesiculation and even gangrene of skin and deeper tissues may occur.

Many enthusiastic accounts of the effects of ultrasonics in a wide variety of diseases have been made, but they are mostly uncritical and uncontrolled.

It is used mainly for its thermal effects and other methods are generally preferable.

DEEP X-RAY THERAPY

In certain traumatic states deep X-ray therapy may have beneficial effect. These are:—

(a) the severely contused and swollen hand and foot;

(b) capsulitis of the shoulder joint, especially if there is a marked degree of joint stiffness associated with calcification of the subacromial bursa or supraspinatus tendon. This form of therapy may be given a trial before considering operative procedures;

(c) established cases of osteoarthritis which have sustained recent injury, especially of the knee, ankle and small joints. Deep X-ray therapy can be combined with other forms of treatment.

INTERFERENTIAL THERAPY

Dr R. P. Goulden has contributed the following.

A new type of electrotherapy has been devised by Dr Hans Nemec.[9] It is a method of producing a wide variety of low frequency currents within the tissues without sensory stimulation of the skin.

Two medium frequency currents of constant intensity are applied separately and simultaneously to the body. The intensity of the combined current increases and decreases rhythmically. In the area within the body where the currents mix, arises a medium frequency current which changes its intensity in a low frequency beat rhythm. These low frequency intensity changes have a stimulatory action inside the body.

By adjusting these low frequency rhythms, different structures are stimulated, and varying therapeutic effects are produced.

There is an analgesic effect which is useful in rendering tissues less tender —so that further physical treatment can be given in comfort, and so that the patient may be able to perform remedial non-weight bearing exercises more easily without pain. This is particularly useful in recent injuries, e.g., sprained knees and ankles. It is also useful in treating painful osteoarthritic joints, and capsulitis of the shoulder.

These low frequency intensity changes can also be used to cause vasodilatation and can be used instead of short wave diathermy without the risk of burning the tissues.

By varying the frequency yet again motor stimulation can also be produced, giving a faradic-like stimulation of muscles. This is particularly useful in reducing oedema of a limb, with the limb in elevation, but is more pleasant for the patient than faradism as there is no sensory stimulation.

Yet another frequency is reputed to stimulate lymphatic drainage. In addition to these the machine can be used for direct current stimulation, interrupted galvanism, and ionisation.

The whole machine is electronically operated. It is small, light and easily portable, and is a valuable addition to the physiotherapists armamentarium.

PROPRIOCEPTIVE NEUROMUSCULAR FACILITATION

Dr A. B. Corrigan has contributed the following.

This is a technique perfected in the United States of America since the end of the war (Kabat, 1952)[10]. Its aim is to stimulate responses in the neuromuscular mechanism and so improve them. This leads to a facilitation of those

patterns of movement which comprise mass movement, this being the basis of normal motor activity. It is axiomatic that the brain knows nothing of individual muscles but only of motion and also that gross patterns of movement are inherent. These inherent motions are produced by synergistic muscles and so an isolated muscle contraction is a learned or acquired function.

The physiological principles upon which the technique is based are derived from Sherrington's Law of Successive Induction. This was realized after the technique had been fully evolved. Originally, muscles were put through their range of movements using maximal resistance against them during repeated contractions. Then, the range of movement was increased to allow of two separate movements of each muscle group—e.g. flexion and extension of foot muscles instead of only flexion. Then stretch was applied to synergistic muscles to obtain greater proprioceptive stimulation. In 1949 a major step forward was made when it was found that an isometric contraction of an agonist, followed by an isometric contraction of the antagonist, results in an increased response by the agonist. Later it was found that spiral and diagonal movements are even more effective.

The two methods used to produce these patterns are:—

1. *Slow reversal:* an active contraction against maximal resistance in the agonist is followed by an isotonic contraction against resistance in the antagonists without relaxation between the two, and

2. *Rhythmic stabilization:* the patient contracts to the strongest part of the range against resistance. Then the patient holds as an equal amount of resistance is applied alternatively to the agonist and antagonist muscles. These alternate isometric contractions result in an increased build up of tension in muscles.

To produce greater proprioceptive stimulation, attention to details is most essential. This involves:—

(i) proper positioning of the limb to produce a stronger contraction in the desired muscle group,

(ii) repeated contractions cause a summation of impulses, besides improving the extensibility and endurance of the muscles.

(iii) manual contact of the physiotherapist,

(iv) stretch is applied to muscle groups,

(v) verbal commands to the patient,

(vi) traction and approximation of joint surfaces increasing proprioceptive discharge from the joint structures,

(vii) maximal resistance is applied to both the isotonic and isometric contractions. This allows of irradiation of impulses from one major muscle group to a weaker group. This implies a sequence of

(viii) *normal timing* which helps in co-ordination. At specific times in a pattern of movement, resistance is increased and irradiation of impulses again ensues.

Part of this technique has of course been used for thirty years in muscle development. Patients have been taught isometric contraction of muscle groups—whilst in plaster casts, attempting to move the enclosed joint, against the resistance of the plaster.

It has also been used for many years in detail for restoration of movement after injury and operations on joints, though one may not have known the theoretical basis for the good results which ensued.

These techniques have been used for some time but the nomenclature is much more recent. The techniques themselves use basic principles and the main use is in overcoming functional inhibition of muscles after injury and in increasing extensibility in injured muscles by giving greater stretch and strength. There is no particular reason for any added confusion in the use of the terminology as advised by Kabat, the important thing being to realize that this is a technique that applies accepted physiological techniques to muscle re-education.

INJECTION THERAPY

There are various injections which may be of great benefit. Their objects are:—

1. To produce an anaesthetic effect on the tissues, in order to stop painful impulses or reflex disturbances, as described by Leriche. The reflex disturbances include protective muscle spasm, which is overcome by local anaesthetics.

2. To help dilute the inflammatory products and to break down thickenings. Initially the fluid injected increases the tissue tension, but it soon diffuses over a wide area, especially if active treatment is given.

3. To introduce specific fluids, such as hydrocortisone and similar compounds, which have an anti-inflammatory action and also increase the permeability of the blood vessels, so that the process of absorption of the effusion takes place more rapidly.

4. To secure vasodilatation, and convert a state of ischaemia of the part into an active hyperaemia.

5. For local manipulatory purposes to sever adherence between two tissue layers.

Injection therapy can, therefore, be used in a recent injury where there is a large extravasation of fluid. In this instance quick absorption is the main object of injection: but as the injury gets older, thickenings form, and the injection is given to break up these, and to help them to disperse and become absorbed.

All injection therapy begins with a local injection of procaine, or xylocaine without adrenalin, usually in 2 per cent solution, to make the subsequent injections painless. About 2-10 ml. are usually required.

After the preparatory injection comes the 'therapeutic principle', which may be:—

1. lactic acid, usually into a joint;
2. salicylic acid-urea solution, also into joints: but often into muscle and soft tissues around the joint;
3. xylocaine in various strengths. This is said to have a more lasting effect than procaine;
4. various weaker solutions of procaine or xylocaine—$\frac{1}{2}$ per cent and 1 per cent;
5. hydrocortisone into the joint, the soft tissues around the joint, or soft tissues generally. In certain conditions, such as tennis elbow and ligamentous strains, hydrocortisone has hastened the recovery period.

Hyaluronidase is frequently added to the fluid injected, as an adjunct to quick absorption. It produces phenomenal effects in recent injuries, especially where there have been large extravasations of blood or where a large haematoma is becoming 'organized'.

Technique: the procedure varies, naturally, in each individual case. After the overlying skin has been thoroughly sterilized, a few mls. of the 2 per cent procaine is injected into the skin over the tender point, and, then, after the area has become anaesthetised, the injection fluid is given through a larger needle at varying depths. When the injection is into a joint, any effusion present is aspirated before the therapeutic fluid is put in. Blood-stained or haemorrhagic synovial effusion fluid is obviously better aspirated out of the joint before hydrocortisone is put in.

Naturally, injection therapy should be combined with physiotherapy, which it greatly enhances. The seeping or spreading of the products of injury is facilitated and the fluid injected breaks up and spreads the effusion and thickenings into areas where the lymphatic and venous circulation

have not been disturbed by the injury, and where they are more easily absorbed.

In large haematomas of recent origin, it is sometimes possible to aspirate the blood through a large bore needle before injecting the fluid. Alternatively the injected fluid may break up the clot, some of which can be aspirated through the needle. In other cases, however, the haematoma has organized and aspiration is impossible: it may have to be expressed through a small incision down through the deep fascia.

The injection may provoke a tissue reaction, with pain of varying degree,

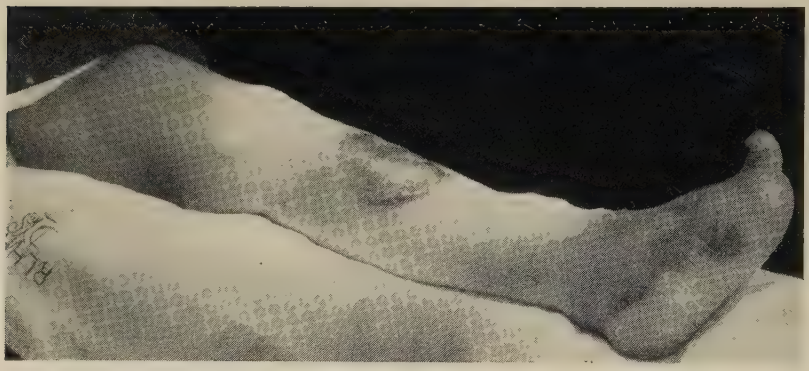

FIG. 8. Extensive contusion bruise of lower leg.

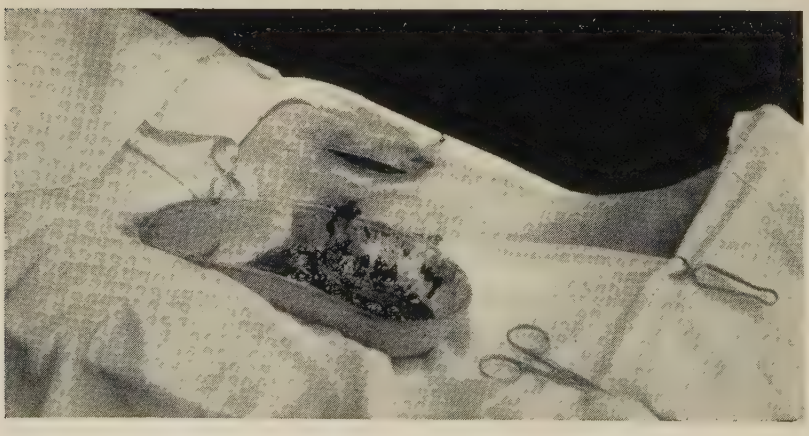

FIG. 9. Extensive opening of the area of contusion, showing quantities of clot removed.

especially the first night. The patient ought to be warned what to expect, and given a combination of a hypnotic and an analgesic such as soneryl gr. $1\frac{1}{2}$, with seconal gr. $1\frac{1}{2}$, to take at night. If he still has pain the next morning he should have a further dose of seconal and two veganin tablets. Occasionally, injections of pethidine or omnopon are necessary to bring him relief.

After traumatic swelling has been broken up and spread by the injection, massage, or faradism are the best methods to hasten absorption.

PAIN IN INJURY

Pain after injury may be very severe. In a recently sprained joint or strain of a muscle, the pain may be so great that an analgesic as strong as morphia may be necessary, for the first night at least.

The causes of pain are:—

1. involvement of a major nerve by the injury;
2. stimulation of sensory nerve endings by the tension of traumatic effusion or actual tearing of the nerve ends in the damage tissues;
3. direct pressure on a nerve root, for example by a displaced fragment of an intervertebral disc. In the case of a nerve root, pain is probably the result of an increase in tension in the root consequent on its displacement by pressure. If the root is actually compressed anaesthesia and paralysis are produced.

In recent injury, nerve filaments in injured muscles and tissues are exposed: traumatic swelling may put direct pressure upon the nerve endings; muscles may go into protective spasm: and all these factors cause pain. The combination may form a vicious circle of nerve irritation, muscle spasm, increased tension and nerve irritation again. To relieve the pain, the object should be the reduction of tissue tension by the spreading or elimination of the traumatic swelling and the reduction of muscular spasm.

The patient is frequently told that many of the methods of treatment of recent injury may cause some reaction and increased pain for a day or so. So long as he has been warned what to expect, and provided the treatment is given cautiously and gently at first, the patient will usually persevere, realizing that his recovery is being accelerated.

In chronic injury, also, treatment may cause definite reaction. A later painful stage, for which the patient must be prepared, comes when the fibrinous portion of the traumatic effusions begins to break up and spread between the muscle layers, or between a bone and the origin of a muscle. Small gritty particles form and these, if pressed may cause exquisite pain.

If an old injury has been neglected, there may be pain, the origin of which is not obvious. Traumatic effusion may have consolidated into thickenings into which fine nerve filaments grow. Movement or massage may make the thickenings oedematous, and the resulting tension causes tenderness and pain.

Tender points tend to form at the convergence of tissue planes, such as the outer side of the erector spinae between the layers of lumbar fascia, and at other points where small cutaneous nerves and, in some cases, blood vessels, pierce the deep fascia to become subcutaneous.

Local pain and loss of movement can also be due to muscle spasm as a result of primary muscle strain or simple muscle cramp. This may be relieved by manipulative stretching, which reduces the spasm and restores normal muscle balance.

These simple methods soon bring relief. As the pain is, in all cases of injury, only a temporary symptom, analgesics should not be withheld. Mild degrees of pain respond to aspirin tablets in the various forms, or veganin; moderate degrees to seconal and pethidine by mouth; severe degrees to injections of omnipon or pethidine. If the patient is emotionally agitated, drugs such as bromo-valerian, luminal or largactil are helpful. Soneryl has some slight analgesic properties as well as hypnotic.

The oral administration of anti-inflammatory enzymes

The dispersal of traumatic swellings can be greatly accelerated by the administration of oral anti-inflammatory enzymes.

The first of these is buccal varidase which consists of a purified and concentrated combination of streptokinase and streptodornase. The streptokinase combines with plasminogen present in the saliva, and is absorbed into the blood stream, via the buccal mucosa. In the presence of inflammatory tissue this activated plasminogen is converted into plasmin. Plasmin acts on tissue fibrin allowing greater permeability of tissue fluids, and also conditions the fibrin for its dissolution and removal from the site of inflammation. The dose is one tablet q.i.d.

The second of these is chymoral which is an oral form of chymotrypsin and trypsin dispensed as enteric coated tablets. These are absorbed from the small intestine. The dose is two tablets q.i.d. and appear to be equally effective as varidase.

REFERENCES

1. Hilton J. (1950). *Rest and Pain.* G. Bell & Sons Ltd., London, 6th edition.
2. Tucker W. E. (1935). *Injuries and their treatment.* Lewis, London, p. 5.
3. Perkins, G. (1953). Robert Jones Lecture 1952 at R. Coll. Surgeons. 'Rest and Movement.' *Journal of Bone & Joint Surgery*, November, Vol. 358, No. 4, p. 521.
4. Platt, Sir H. (1955). *Journal of the Royal Army Medical Corps*, January edition, p. 43.
5. Cyriax, J. (1959). *Textbook of Orthopaedic Medicine*, Vol. II.
6. Mennell, James B. (1949). *The Science & Art of Joint Manipulation.* J. & A. Churchill Ltd., London.
7. Seddon, H. J. (1949). 'War Injuries of Peripheral Nerves.' *Brit. Journal of Surgery.* War Surgery Supplement No. 2, p. 325.
8. Smart, Morton. (1933). *Principles of Treatment of Muscles and Joints by graduated Muscle Contractions.* London, Chapter 12.
9. Nemec, H. (1959.) 'Interferential Therapy: a new approach in physical medicine.' *British Journal of Physiotherapy.* December. Extract of a translation of book by Hans Nemec.
10. Kabat, H. (1952.) *Archives of Physical Medicine*, 33-521.

4

THE CLASSIFICATION OF INJURIES AND THEIR TREATMENT

including

'Injuries to Arteries' by Sol Cohen, F.R.C.S.

In the last chapter modern therapeutic principles and methods were discussed; the present chapter is to be devoted to specific injuries and how to treat them.

A suitable classification of these injuries is as follows:—

1. Those involving skin
2. Those to muscles and tendons
3. Those to joints
4. Those to bursae
5. Those to bones
6. Those to nerves
7. Those to arteries

Injuries to bones are considered in detail in other chapters. Injuries to the skin and nerves are very specialised subjects, the details of which are beyond the scope of this book. They are considered here only in outline. The others, which form the vast majority of athletic injuries, are considered in some detail. Mr Sol Cohen has contributed a useful chapter on vascular lesions.

INJURIES TO THE SKIN

Even in the athlete the skin is the body's first line of defence. When breached, bacteria can enter the tissues, multiply and cause local or general infection. The aim of treatment, therefore, is to seal the breach as soon as possible; that is to say, to get rid of the surface wound as soon as possible.

The physical barrier to bacterial invasion is thus re-established; but it is also of the utmost importance that the bacteria which have already entered should not be allowed to prosper. When the wound is treated, therefore, dirt and devitalised tissues, which provide a favourable culture medium must be removed. Proper surgical cleansing gives the best chance of healing without

infection and avoids the development of an adherent tender scar which can be both disfiguring and disabling.

During his examination the surgeon must estimate not only the degree of skin damage, but also that of the deeper structure. If he has any doubt how far the wound extends he ought to make a proper investigation under full anaesthesia.

Skin damage may be either a clean or a ragged incision or a contusion, with or without loss of the superficial layer, or the whole skin thickness. The treatment of each lesion depends on the time that has elapsed since the injury.

Recent cuts or incisions: If the wound is a simple cut primary suture must be undertaken at once after cleansing it. A ragged skin wound must be sterilised and all dead and devitalised tissue removed. Suture or skin grafting can usually be undertaken as a primary procedure, especially if helped by antibiotics. These procedures are extremely important in relation to wounds around joints such as the knee when there is an associated internal derangement. Operation for such a complication may be delayed for weeks by bad primary treatment of an associated skin wound.

Recent contusions: Although there may be no actual break in the surface, the superficial and deep layers of the skin may be injured and cover a haematoma in the subcutaneous tissues. This often produces tension which results in decreased viability of the superficial layers. If the area of contusion is large, it is wise to examine the wound in a proper operating theatre under a general anaesthetic, so that the surgeon can make an unhurried inspection and carry out whatever treatment proves necessary.

It is essential to remove any obviously dead tissue. It may be possible to replace lost or excised skin at the same operation by a split skin graft: or, it may be necessary to wait until the non-viability of the underlying tissue is beyond any doubt. Such an injury may need systemic chemotherapy or antibiotics, and local daily dressing with solutions such as Eusol.

If there is a haematoma, the blood should be evacuated and devitalised tissue removed. The negative and positive phases of healing have already been discussed, and this is an example of how, through surgical intervention, the negative phase may be cut short or completely eliminated and the positive phase of healing encouraged.

Contusion seen some time after injury: Even today one sometimes sees a patient with a neglected surface contusion which involves the subcutaneous tissues. Over the area of injury there may be a thick black eschar which signifies that the involved skin is dead. At this stage operative removal of the

necrosed skin and evacuation of the haematoma will improve the chances of survival of the underlying tissues and promote quicker healing.

At the first examination in some cases a granulating healing wound is found which, after suitable preparations, must be covered with a skin graft. If the granulations are infected the wound will need a period of cleansing before a graft can be applied successfully. This usually implies currettage of granulations, bacteriological tests, antibiotics and daily dressings. The type of graft used depends on the state and extent of the wound. Split-skin grafts are usually employed, and grafts of various sizes and thickness are used in different circumstances.

Inadequately treated injuries more than two months old may develop a contracted scar adherent to deep structures; this may respond to a conservative treatment or may require operation. The details of treatment of such scars is considered briefly in Chapter 9, page 457.

INJURIES TO MUSCLES AND TENDONS

Injuries to muscles and tendons may be classified as follows:—

(a) contusions, superficial or deep:
(b) a strain of a muscle or tendon:
(c) rupture of a few fibres:
(d) partial rupture of a muscle or tendon:
(e) complete rupture of a muscle or tendon:
(f) avulsion of the tendon of origin or insertion with flake of bone:
(g) tendonitis or tenovaginitis—acute:

A muscle may be injured anywhere along its length, especially if it is subjected to direct violence. In indirect trauma also any part of the muscle or tendon may be involved. Strains or tears are most common, however, either at the tendon of origin or insertion, at the musculo-tendinous junction, or where the tendon changes direction in its course.

Contusions

The degree of damage naturally depends on the force of the blow: it may be slight or severe enough to lead to the death of muscle fibres. A haematoma nearly always forms. If the tendon is involved fluid collects between the tendon and its sheath, and acute tenovaginitis develops with pain and crepitus on movement.

In a minor contusion on the surface of the muscle, home treatment with physiotherapy and graduated active exercises often produce a cure in a few

days. Some form of removable protection and support, such as a crepe bandage reinforced by a figure of eight elastoplast may be helpful when the patient begins vigorous exercise.

If the contusion is deep in the substance of the muscle, there are two possible approaches. The first is relatively conservative: the second is more drastic—an attempt is made to find the bleeding point and to deal with it by ligature, coagulation or packing.

Conservative treatment allows the amateur athlete to carry out his work; if search for the bleeding point is carried out by operative means the patient may be incapacitated for at least a fortnight. Some years ago, for example, an athlete who is an actress sustained a contusion with the formation of a large haematoma in the right buttock. This was treated conservatively: under local anaesthesia a small incision was made over the swelling. She was able to continue her important role in a London play during the period of healing. The second, more drastic method should be seriously considered for the professional athlete, particularly in those cases of injury of the deep surface of the thigh muscles where bleeding is often associated with tearing of the subperiosteal and parosteal blood vessels along the shaft of the femur.

If the haematoma is a large, fairly superficial one, it saves time to evacuate it either by aspiration in the first few hours, or if this is not possible, owing to clotting, by expressing it through a small incision under local anaesthesia. A solution of urea and salicylic acid with hyaluronidase is injected to help absorb the remains of the haematoma, and physiotherapy is begun at once. Even a severe contusion may become completely symptom free in two to four weeks by such active treatment.

A muscle swelling after an apparently trivial blow may contain several litres of blood and traumatic effusion fluid, especially if an artery or vein is partially torn deep in the substance of a muscle. For example, one evening an international squash racket player presented himself for examination. Five hours previously he had given himself what appeared to be a trivial injury: he had bumped himself against the back wall of the squash court. At the time he was not aware of any damage but in his bath an hour later he felt the buttock becoming stiff and painful. It gradually swelled up until at 9 p.m., when he was seen, the buttock had swollen to the size of a football. The haematoma was pressing on the sciatic nerve and he had acute sciatic irritation with pain right down to the heel. He was immediately admitted to hospital and under local anaesthesia an incision 1 in. in length was made just at the point of maximum swelling and tenderness. There was no attempt to locate the bleeding point, but as the incision cut through the deep fascia, many ccs. of serum seeped up through the tissues. The dressings had to be changed twice daily for

the first two days. He was kept in bed for a week and given exercises and physiotherapy; and the serum continued to seep through. At the end of the week there was a marked improvement and he was able at the end of a fortnight to start playing squash rackets again. In exactly four weeks from the date of injury he was able to play in an International Squash Rackets final. Subsequently he has played squash rackets and real tennis without trouble and there is not the slightest suggestion of an arterio-venous aneurism. Similar cases have been reported in which the bleeding point was sought and it proved necessary to tie the common iliac artery in order to check the haemorrhage.

In some cases however the contusion of the muscle is situated close to the underlying bony structures and must be treated really seriously, lest myositis ossificans should develop. This is common following injuries of the elbow joint, quadriceps and adductors of the thigh. Aspiration under local anaesthetic should be performed at once and urea and salicylic acid solution with hyaluronidase is instilled deep into the muscle swelling. The area should be rested from all strain, and suitable home treatment and physiotherapy started. If at the end of three days a deep seated swelling which may be visible on a special soft tissue radiograph, has not subsided, it must be explored under a general anaesthetic. With the use of a sucker and diathermy the operation field is left absolutely dry and 5 mls. of a saline solution containing 2,000 units of hyaluronidase is left in the area. Graduated exercises, support of the thigh and knee in split plaster of Paris splint and physiotherapy are commenced as soon as the patient has recovered from the operation. The patient is encouraged to practice active flexion of the knee with the plaster casing removed and the weight of the lower leg taken by the heel on the bed. By these means complete recovery is possible within three weeks.

Frequently, however, this type of injury is seen at the end of three to six weeks when myositis ossificans can already be detected in the radiograph. The treatment of this condition will be considered later in injury of the thigh muscles.

Strain of a muscle or rupture of a few fibres

These two degrees of injury can be considered together as the treatment is the same.

The injured muscle is protected, supported and rested from strain. Small injections (as for muscle contusion) may be given. Physiotherapy and home treatment accelerate recovery.

Provided a large haematoma does not form, gentle active exercise can be started in a few days and there should be complete recovery in a fortnight.

Partial rupture of a muscle

It used to be stated that muscles partially ruptured never recovered enough to allow the athlete to take part in vigorous athletics again in the same season. This was chiefly because the patient simply rested without active treatment. When he resumed hard exercise the tear would recur, either because adhesions had formed in the muscle or its surrounding tissue, or because there was a weak scar or because of loss of tone in the damaged muscles. Today, by a regime of active treatment, we expect complete recovery in four to six weeks. According to the severity of the tear the injury will take two to three weeks to become symptom free. Injection therapy, varidase or similar pills by mouth, daily physiotherapy and the use of twice daily home treatment are essential to accomplish this. If the muscle is likely to be put to vigorous use, it is best to insist on a further fortnight's building up by graduated exercise, or the tear may recur.

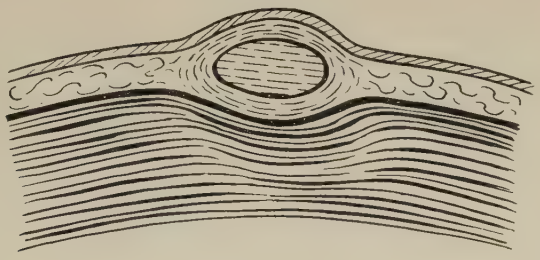

FIG. 1. Subcutaneous encystic swelling as the result of contusion of the surface of the muscle.

Remarkable results have sometimes been credited to a particular type of treatment: but these are usually cures of very light tears or cases in which there has been a previous history of a muscle injury and the recent injury has torn not the muscle but old adhesions. After such an episode, although the muscle is temporarily sore, any reputable form of physiotherapy will produce a rapid cure.

Certain muscles, the hamstrings for example, may not recover full extensibility even after three weeks. It may then be wise to manipulate under a general anaesthetic in order to stretch them fully and procure complete recovery. After the stretching under general anaesthetic the muscle must be given time to recover before it is subjected to the stresses and strains of vigorous sport.

Some surgeons advocate the injection of procaine in muscular strains and tears, encouraging the patient to start active weight bearing exercises im-

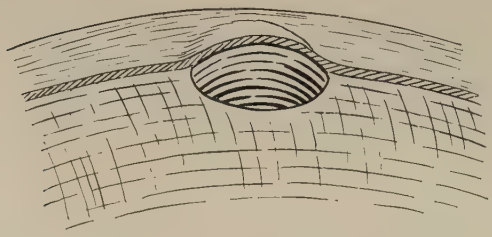

FIG. 2. Muscle swelling due to an encysted haematoma just under muscular sheath.

mediately. This form of treatment may work occasionally, but the muscle often tears again and recovery is delayed. The patient may even suffer permanent damage.

A more rational line of treatment is to disperse the effusion, at the same time resting the part from strain. Meanwhile tone and power are restored by gentle remedial exercises, which are carefully graduated until the full extensiability, flexibility and power return.

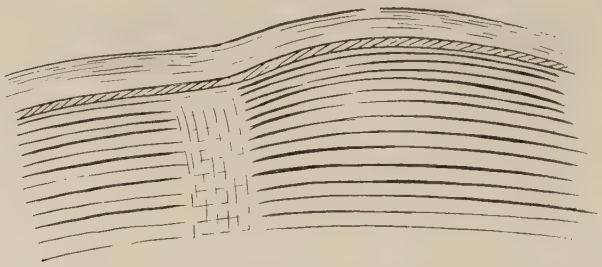

FIG. 3. Muscle swelling due to compensatory hypertrophy of muscle fibres of one part of the muscle owing to rupture of the muscle in its course.

Complete rupture of a muscle or tendon

So long as the ruptured ends are not distracted by active contractions, healing of the muscle is possible: but in athletes unless there are special contraindications, ruptured muscles which bear weight should be sutured. The exception to this rule is the rupture of the insertion of the rectus femoris into the main mass of the quadriceps. With active treatment any small effusion is eliminated and the other muscles of the group take on the function of this muscle, with no loss of power.

Some tendons, such as the long head of the biceps brachii, need not be sutured in the older patient, provided active treatment including rest, support, graduated exercises, physiotherapy and home treatment are commenced immediately. In weight-bearing tendons, such as the tendon Achilles, the torn ends usually retract, possibly within the paratenon; and this may be most misleading. It is difficult to obtain a hundred per cent recovery without operation in such cases. Satisfactory suture possibly with fascial graft reinforcement may be successfully undertaken even months after the injury. A fascial graft from the ilio-tibial band may be employed to reinforce the suture of the torn ends, especially in athletes and in patients in whom it is difficult to obtain a good strong approximation of the ruptured ends. After operation the limb is splinted in plaster, in a position which relaxes the torn muscle. Gentle, active, isometric contraction of the sutured muscle is begun at once. After a month the plaster cast may be split to form a bivalve splint, which may be taken off for non-weight bearing exercises for the next fortnight, but kept on for walking. After six weeks, support by gamgee and bandage is usually sufficient. Home treatment and physiotherapy aid rapid recovery. A 100 per cent functional result may be anticipated within six months. In the middle aged and elderly athlete, whose requirements are usually limited, a sufficiently satisfactory result may be achieved without operation if the torn ends of the tendo achilles can be felt to come together when the knee is flexed and the ankle plantar flexed. The leg is immobilised in plaster in this position for at least six weeks. During this period, active isometric exercises are carried out. At six weeks the after treatment is the same as that for the tendon which has been sutured.

Avulsion of the tendon of origin or insertion with a flake of bone

Examples of this condition are avulsion of the insertions of the supraspinatus, triceps or long extensors of the fingers (mallet finger) or of the tendon of origin of the adductor longus from the pubic ramus.

In adolescents avulsion of epiphyses is relatively common, associated with a sudden muscular contraction, e.g. anterior inferior iliac spine avulsed by pull of rectus femoris and lesser trochanter by psoas.

The treatment depends on whether or not there is marked displacement of the avulsed tendon. It can often be apposed to its place of origin by manoeuvring the limb, for example by abducting the arm in supraspinatus tendon avulsions: but if the surgeon has any doubt of a perfect reposition he should expose the tendon and suture it back in position. In either event, graduated active use is started as soon as possible. Even if the injured part is held in plaster, isometric exercises are started at once and the torn muscle fibres

gently stimulated by faradism. Plaster support should be maintained for four to six weeks after operation, but full recovery may be expected in eight to twelve weeks.

Tendonitis or tenovaginitis

Inflammation of a tendon or its sheath may be the result of a direct injury, such as a blow, or it can occur indirectly associated with a sprain of a joint. Active treatment in the form of support, physiotherapy and home treatment will generally effect a cure within a few days.

If, however, the condition has been left untreated and allowed to become chronic with the formation of thickenings and adhesions, it may take a long time to cure. Conservative treatment may be supplemented by injection of hydrocortisone but if the tendon is constricted within a thickened sheath as in stenosing tenovaginitis, it is sometimes much quicker to free the tendon by exposing and incising the sheath.

There is a type of tenovaginitis which follows over-use of the muscles. Strains under certain circumstances, such as in the presence of a toxic focus or some metabolic disorder, may produce the condition, either acutely or subacutely. The marked crepitus felt on moving the tendon within its sheath gives the condition its name of tenovaginitis crepitans. Any septic focus must be eliminated. Local support and protection is required so that the movement, which is usually essential to the patient's occupation, does not produce strain. This does not necessarily mean that he must stop his occupation, but the support should check the extremes of movement. He should carry out home treatment and receive organised physiotherapy, preferably deep friction massage and interferential therapy. Recovery normally takes two or three weeks. If the only treatment is rest, the condition settles but is inclined to leave the tendon sheath or paratenon thickened and the muscle weakened and atonic. Return to activity under these circumstances, unless it is carefully graduated and combined with active treatment, is apt to bring about a recurrence. Before the thickened sheath and tendon return to normal, marked swelling and increased tenderness sometimes develop, but the treatment must, nevertheless, go on. During recovery, small sago-like particles varying in size from a pinhead to a pea, break off. These can be made to run up and down the tendon sheath as they are lying free inside. They are gradually absorbed, but if they are trapped by the massaging finger they are exquisitely tender. Patients have described the sensation as one of fine, broken glass in the tendon sheath.

INJURIES TO JOINTS

Joint injuries may be classified as follows:—

(a) Sprains
(b) Internal derangements
(c) Dislocations
(d) Fractures involving the articular surfaces
(e) Miscellaneous group

Sprains

The degree of joint sprain will depend on the force employed: for example, if the patient turns his ankle over as he walks, the chances are that he will sustain a mild simple sprain of the lateral collateral ligament, particularly the anterior fibres. This is because the foot is adducted and inverted, and the ligament is put on the stretch, so that the outer side of the joint bears the brunt of the injury. This mild degree of sprain involves only the ligament, and the underlying synovial membrane is not irritated. There is localised swelling only over the injured ligament and slight pain when it is stretched.

The treatment for a ligamentous sprain is:—Modified exercises with the ligament supported by some type of removable support—a felt pad over the tender point and a firm crepe bandage is usually sufficient: weight bearing in moderation: home treatment: physiotherapy, in particular massage and gentle manipulation which are carried out after the joint has been sprayed with ethyl chloride: and injection therapy if the patient needs to use the joint for vigorous exercise at the earliest possible moment. With attention to detail, the mild degree of joint strain should be completely recovered within a week. The period may even be reduced to one or two days.

If the force is great, as when a runner ricks over his ankle at speed, not only is the ligament sprained or ruptured, but the underlying synovial membrane is lacerated. The result is a moderate or severe degree of synovitis associated with a tear or rupture of the lateral collateral ligament. There is likely to be marked swelling not only of the torn ligament, but of the whole joint.

A severe degree of joint sprain may be associated with a dislocation, articular cartilage erosion or a fracture, and the treatment of these will be considered later in the chapter.

Treatment of moderate sprain: The effusion should be aspirated under local anaesthesia, and hydrocortisone 1·5 ml with hyaluronidase, should be injected into the torn ligament and joint. These should be support and protec-

tion; rest from weight bearing if the injury is to the hip, leg or ankle; home treatment; physiotherapy; postural drainage at regular intervals.

Treatment of severe sprain: Treatment should be conservative unless there are ideal circumstances for operative measures. If radiographic examination reveals opening of the joint on the same side as the ligamentous injury, exploration of the joint surface and resuturing of the ligament are more certain to effect a cure in a reasonable time—six to eight weeks.

If conservative measures are adopted, treatment is similar to that for moderate sprain, except that more complete support is necessary; and this is best achieved by means of a removable plaster of Paris cast. Postural drainage is of the greatest importance and weight bearing is not allowed until the joint effusion has subsided, which is usually in about seven days if concentrated appropriate home treatment and physiotherapy have been employed.

Patients who have had a moderate sprain should return to full activity use in two weeks; those who have had a severe sprain and been treated without operation, in three to six weeks. If the joint has been operated on, it is placed in a plaster of Paris cast for ten days, and the patient is given isometric exercises, faradism (if available) and postural drainage. After ten days, by which time the stitches have been removed, the plaster is discarded in favour of a removable plaster cast which is taken off for non-weight bearing, or non-strain exercises, home treatment and physiotherapy.

No weight bearing or strain are allowed for a further three weeks but then movements, carefully graduated, are encouraged; so that in six weeks there should be full range. The patient, if he is an athlete, can start vigorous training and be fully fit in ten to twelve weeks.

Internal derangement

At the time of a sprain of a joint, one of the intra-articular structure or part of the articular cartilages may be injured and possibly torn. There is usually a severe reaction with effusion, either synovial or haemorrhagic, and there may be locking of the joint.

The muscles are likely to go into protective spasm, which may itself prevent reduction of any torn or contused intra-articular structure. The structures liable to such injuries are the menisci of the knee and temperomandibular joints. Forcible manipulation under anaesthesia may have the effect of further crushing the meniscus, gentle manipulation, on the other hand, with some traction, may reduce the torn meniscus, either directly, or indirectly by reducing muscle spasm. If reduction is successful, the joint should be treated on the lines of a moderate degree of sprain; that is to say, it should be given support, graduates exercises, physiotherapy and home

treatment. If the internal derangement keeps recurring, operative removal of the torn meniscus is the only course likely to give permanent recovery.

Dislocation

A dislocation occurs when the opposing articular cartilaginous surfaces are so deranged that their normal relations are completely distorted, perhaps because a ligament has ruptured, or because the bones are fractured. There is usually a marked extravasation of traumatic effusion into the tissues outside the joint capsule, if this has been torn. At this stage it is imperative to note whether there is any concomitant injury or involvement of the nerves and blood vessels associated with the joint dislocated. This examination should invariably be performed, but particularly upon all cases of severe sprain, dislocation and fracture involving the articular surfaces. Reduction must be performed with the most scrupulous care so as to avoid further damage, ideally in a sterile theatre. If necessary the joint and surrounding structures are decompressed. If the reduction is stable, or if it has been achieved by operation and remains stable after this, the joint is placed in some type of removable support and the gentlest of home treatment and physiotherapy commenced at once. By these methods full recovery is effected in four to six weeks.

If the joint is unstable after manipulative reduction, open reduction and suture of the torn capsule and ligaments is the best treatment: but if operation is impossible owing to unfavourable circumstances, it may be necessary to maintain the reduction by closed plaster which has to be worn for at least a month. During this time isometric movements and faradic contractions through windows cut in the plaster are carried out daily.

At the end of a month the plaster of Paris splint is split and is left off for increasing periods whilst home treatment and physiotherapy are given. Full recovery should take place in three months.

Some dislocations are liable to recur because the capsule and the rim of cartilage surrounding the articular surface are ruptured, for example, detachment of the anterior glenoid which follows some dislocations of the shoulder joint. This type of injury may be due to a combination of direct and indirect trauma, being the result of a blow on the outer side of the shoulder at the same time as a fall on the elbow. The anterior capsule and the gleno-humeral ligaments are torn from their attachment to the glenoid rim and sometimes the cartilaginous rim itself is detached. There is now general agreement that the soft tissues should be the main concern of the treatment, with the aim of preventing traumatic capsulitis. If a dislocation of the shoulder recurs more than three times, as it certainly will if the glenoid rim is detached, operation

is the only method of achieving a cure. The surgeon should advise the patient to submit to operative repair at once, so that no further time should be wasted.

Fractures involving joints

These fall into two groups: those which are stable and those which are unstable.

It is essential to aim at exact reduction of the fractured articular surfaces. Sometimes it is extremely difficult to hold a small fragment in exact alignment, but large fragments can be fixed by screws, nails or stick grafts of cortical bone. Occasionally there is so much comminution that the best one can do is to set the various pieces into approximately the right position by a period of continuous traction and a well moulded plaster.

In some fractures involving joints, the articular cartilage is ploughed up, as it were, and unless it is replaced it usually dies and forms a loose body in the joint. Pridie and Smillie have shown that large pieces of knee joint cartilage can be replaced, provided their bed has been freshened by the removal of the underlying avascular subchondral bone, which may have become sclerosed.

Restoration of joint movement is encouraged by active isometric contractions of the overlying muscles while consolidation is taking place. In some cases it is possible to cut windows in the controlling plaster without disturbing the continuity of the fractured surface, in order to give daily faradism. A certain amount of joint stiffness is almost inevitable; but as soon as the patient is allowed to move the joint, he can, by perserverance with home treatment and periodic courses of physiotherapy, gradually restore the range of movement. In some cases, periodic injections of hydrocortisone will help to accelerate recovery. Immobilisation in the closed plaster may be necessary for as long as eight weeks, but with active home treatment, physiotherapy and injections an excellent result can often be obtained within the year.

Miscellaneous group of joint conditions

Included under this heading are chondro-malacia patellae, osteochondritis dessicans and the various types of chondroepiphysitis.

Although there is not sufficient evidence to state dogmatically that these conditions are entirely due to trauma, a particular movement and possibly injury sometimes appear to be the aggravating cause. Certain of these conditions will be considered in detail later.

INJURIES TO BURSAE

Many bursae, owing to their exposed position, are liable to direct injury,

particularly the prepatellar and olecranon bursae. Other bursae such as those at the insertion of the muscles around the shoulder, hip and knee joints become inflamed through indirect injuries. Such inflammation may follow excessive use of a muscle or irritation of the bursa by the products of traumatic effusion from tissue contusion; for example, Achilles tendon bursitis may follow a tear of the calf muscles. Morton Smart described how bursitis may develop from the irritative effect of a slack atonic muscle and how it may be cured by improving the muscle tone. Vigorous exercise of an unusual sort is often the beginning of a lesion of this type: for example subacromial bursitis as the result of excessive bowling at cricket or pitching at baseball.

The inflammation of a bursa may be acute, subacute or chronic.

If it is acute and due to a direct blow a haemorrhagic effusion may develop in the bursa and should be aspirated under local anaesthesia. Physiotherapy (anodal galvanism and massage) with home treatment, protection and support will effect a cure in a short time.

If the inflammation becomes subacute or chronic, the walls of the bursal sac become markedly thickened and the sac may contain particles of inspissated material described as 'melon seed bodies'.

Conservative treatment in the form of injections, massage, short wave or anodal galvanism may effect a cure in time, but often operative excision of the bursa is a quicker and more satisfactory method.

If recovery is slow or if the inflammation recurs one should look carefully for a hidden focus of infection. Occasionally the walls of the sub-acromial bursa become calcified, and if irritated by over use, cause acute pain. The whole sac becomes the site of inflammation which spreads to the neighbouring structures. The pain can be so acute that sleep is impossible and the slightest movement causes agony. It is worth trying conservative treatment—rest, butazolidin by mouth, gentle physiotherapy and faradic contraction for two days: if pain is not alleviated, deep X-ray therapy daily for four days sometimes works: but if excruciating pain persists, operative removal of the bursa and the acromion process may be the only satisfactory measure.

CONTUSION OF BONE AND CARTILAGE

Bones and cartilage which are near the surface, such as the subcutaneous border of the tibia and the cartilaginous extremity of the nose, are especially prone to injury. The resulting traumatic effusion varies from a slight ecchymosis over the bone or cartilage to a definite subperiosteal or subchondral haematoma. The area of contusion is exquisitely tender to the slightest pressure. If left to itself the traumatic effusion gradually tends to absorb and

spread, leaving behind areas of painful granulation tissue. If the limb is exercised, it aches and swells. The effusion may not resolve entirely and there may be left behind a circumscribed organising haematoma, which may remain for months or years. The area of tenderness will spread owing to seepage of the fluid into surrounding tissues.

If the swelling is large, the blood and fluid should be evacuated under local anaesthesia; but in women or children, the expression of large diffuse haematoma is such a painful process that it is best done under general anaesthesia.

Strict asepsis is essential, and if there is any possibility of infection, antibiotics may be exhibited. After most of the contents of the haematoma have been expressed, further absorption of the remnants can be accelerated by leaving in the wound 5 mls. of $\frac{1}{2}$ per cent procaine or xylocaine to which has been added 1,000 units of hyaluronidase. Varidase or Chymoral given orally assists rapid absorption. Daily physiotherapy, home treatment, pressure dressing and postural drainage by elevation of the limb must play their part.

Footballers and cricketers are particularly prone to the formation of haematomata near the subcutaneous border of the tibia. Weeks, even months can be saved by the timely active treatment of these conditions, and even the biggest haematoma can be made symptom-free within three or four weeks. Patients who have had no specific treatment for this type of injury have been found to have painful swellings even months or years after injury. For example, three years after a blow on the upper outer side of the tibia a man still had a swelling in the upper quarter of the anterior compartment of the leg. The swelling was the size of an orange; it was tense and tender to pressure; it caused a continuous ache; and a diagnosis of sarcoma seemed possible. At operation it was found to consist of a fibrous outer covering containing altered blood and fatty tissue in the centre. There was evidence in the surrounding tissues that blood had tracked and seeped in the anterior compartment of the leg right down to just above the ankle joint. A month after operation the patient was symptom free.

INJURIES TO THE NERVES

The main purposes of this survey of nerve injuries is to stress the importance of testing for the presence of a possible nerve involvement in every case of injury immediately after its occurrence. If this examination is omitted or skimped, not only may signs which are of the greatest value in diagnosis and prognosis be missed, but the consequence for the patient may be most serious. Then, if at a later date typical signs and symptoms of nerve damage appear, a proper assessment of the lesion may be difficult. In addition to the moral—

and the legal—considerations, a major clinical problem arises:—are the effects of nerve damage still developing or has the recovery started? The subsequent treatment naturally depends on the answer.

Special investigations and examinations upon patients in whom nerve involvement is suspected should include:—

(a) Review of the history of the injury. Has there been a wound through which the nerve could have been severed? Is there a fracture which may have damaged a nerve?

(b) Attention to the attitude of the limb. It may adopt a characteristic position at once, owing to the paralysis of certain muscles; for example, the wrist drop in radial nerve palsy or the foot drop in peroneal nerve palsy. Once muscle imbalance is established the limb adopts a characteristic position, for example the ape or flat hand in an established case of median nerve palsy and the griffin hand in ulnar nerve palsy.

(c) Tests of certain reflexes. The entire reflex arc must be intact, otherwise there will be alteration in the reflexes. When peripheral nerve damage occurs, certain reflexes may disappear.

(d) Assessment of condition of the muscles. Are any of them wasted, or do they feel as if they have lost their tone? If so, how much has their power been affected?

(e) Tests of alteration in skin sensation to pain (pin prick), temperature (hot and cold water), touch (cotton wool). There may be hypoaesthesia (impaired sensation), or hyperaesthesia (increased sensitivity).

(f) Observations on the condition of the skin, nails and circulation generally. Are there trophic and vasomotor disturbances? Is the skin cold and dry, with cracks, or is there swelling with heat?

(g) Measurement of the electrical responses of muscles. Whether the Reaction of Degeneration (R.D.) be present or not.

Electrodiagnosis

Wynn-Parry[1] has given a most comprehensive survey of the importance of Electrodiagnosis. The substance of his article stresses the following:—

Electrodiagnosis is the study of the reaction of voluntary muscles and nerves to electrical stimuli, by:—

(a) Tests for nerve conduction and
(b) Intensity duration curves.

It embraces also the analysis of the electrical activity produced in these tissues by means of electromyography.

1. Nerve Conduction Test: By stimulating a motor nerve where it appears superficially by means of a surface electrode, and observing the contraction of the muscles innervated by the nerve.

2. Intensity Duration Curves: These are a study of the reaction of voluntary muscles and nerves to electrical stimuli of known strength and known duration. They are graphic expressions of the excitability of neuromuscular tissues.

The curves are obtained by plotting the intensity of electrical stimulus required to produce a minimal muscular contraction against the duration of that stimulus.

Nervous tissue is more excitable than muscle tissue, therefore an inner-

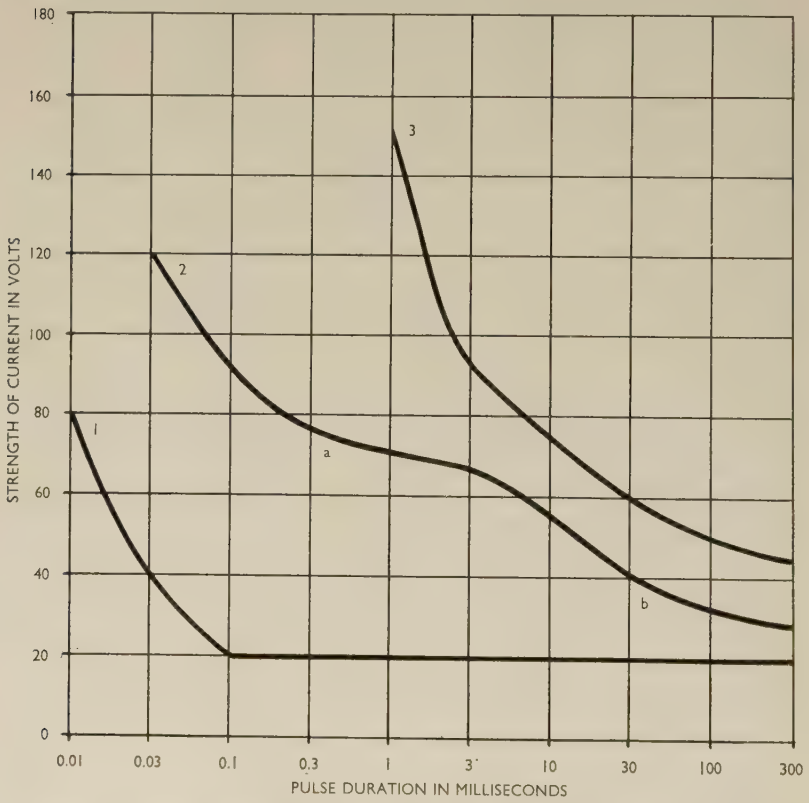

FIG. 4. Pulse duration in milliseconds. Strength duration curves of (1) normal, (2) partially innervated muscle, (3) denervated muscle: (a) represents the innervated part and (b) represents the denervated part of the muscle.

vated muscle is stimulated indirectly through its intra-muscular nerve branches. If the muscle is denervated then the stimulus acts directly on the muscle fibres.

Thus three types of graph can be obtained which represent the excitability of:—

1. Normal muscle.
2. A partial nerve lesion which may be obtained during the process of denervation, or during renervation. Thus if a partial lesion is present, serial graphs at fortnightly intervals should be performed to observe progress.
3. Complete denervation.

It will readily be seen that these curves are of great use in diagnosis. They are also useful in prognosis in a particular lesion—as when renervation proceeds—the kink in curve II gradually flattens out and the curve approaches the normal Curve I.

3. Electromyography: The activity of muscle or nerve fibres is accompanied by changes in cell membranes and consequently the production of action potentials. Electromyography is the study of the action potentials of muscle fibres since these predominate.

The functioning 'motor unit' of voluntary muscle consists of the anterior horn cell, its branching axons, and the 150 or so muscle fibres it supplies.

For clinical work an electromyograph is carried out by a concentric needle electrode which is inserted into the muscle to be tested. The action potentials are picked up by the needle and led to an amplifier and then fed into a cathode ray tube where they can be observed on a screen. They can also be led into a loud speaker. Certain electrical responses give characteristic sounds and can be recognised by ear alone.

Normal healthy muscle at rest is electrically silent. On voluntary contraction the smallest detectable potential is that of the motor unit, as all the muscle fibres supplied by one anterior horn cell will contract at once. Contraction of a single muscle fibre can only occur under abnormal conditions.

In pathological conditions any spontaneous activity in the resting muscle is observed. The size of the motor unit potentials on minimum voluntary contraction is observed and finally the interference pattern on maximum voluntary contraction is observed.

From an analysis of the nerve condition, the intensity duration curve and the electromyographic findings, it is possible to localise the lesion to one part of the motor unit, viz:

(a) Myelopathic in the A.H. cell

(b) Proximal neuropathic in the axon
(c) Distal neuropathic in the intramuscular branches
(d) Myopathic in the muscle fibres themselves

Seddon[2] classifies nerve injuries into:—

1. Neurotmesis (syndrome of complete interruption).

The effective continuity of the nerve has been broken; muscular action ceases, wasting is soon apparent and there is complete reaction of degeneration in the muscles. Lesions of this type are often associated with open wounds like compound fractures and gunshot wounds. Occasionally after a sudden severe stretching of a nerve subsequent intraneural haemorrhage and fibrosis prevents spontaneous regeneration of the axons so that although the physical continuity of the nerve is not interrupted there is a bar to regenera-

FIG. 5. Neurotmesis.

tion. As an example, paralysis of the deltoid following a dislocation of the shoulder may be quoted. In such circumstances the circumflex nerve is stretched and recovery of function in the deltoid depends on the reversability of the changes that take place in the nerve. Traction injuries cause severe disability and are not amenable to operative intervention. Owing to the extensive nature of the lesion it is impossible to close the gap by direct suture after resection of damaged nerve tissue or even by nerve grating.

2. Axonotmesis (syndrome of lesion in continuity).

In such a lesion the stroma of the nerve remains intact although there is disruption of the axons. Such lesions occur most commonly in association

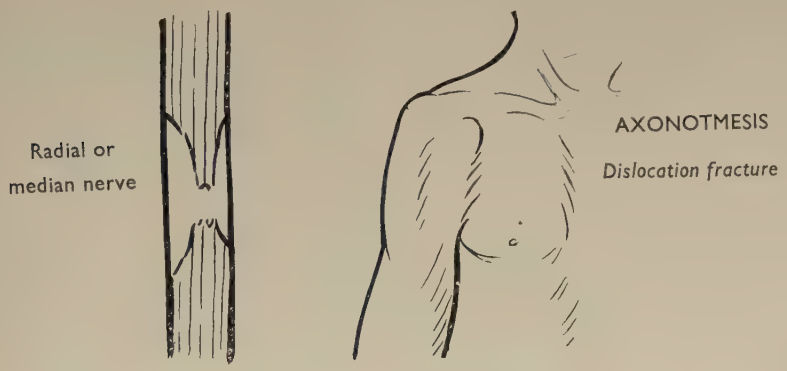

FIG. 6. Axonotmesis.

with closed fractures and can be compared to phrenic nerve crush by an artery forceps. Full spontaneous recovery occurs. The axons remaining in their respective sheaths ensuring that it is perfect. Clinically, however, there is no means of distinguishing between neurotmesis and axonotmesis in the early stages apart from operative intervention. In both instances there is reaction of degeneration, muscle wasting, and complete motor and sensory paralysis.

3. Neurapraxia (non-degenerative nerve lesion).

A typical example of this is the temporary paralysis of the radial nerve caused by the pressure on the back of a chair (Saturday night palsy) or the lateral peroneal nerve palsy from wearing tight breeches. There is a paralysis but this is due to a local demyelination of the fibres and not to degeneration of the axons. The electrical reactions when examined 21 days after injury are quite normal. The patient can be reassured that full recovery will occur rapidly.

Treatment

The nature of the treatment must obviously depend on the type of injury and whether there is an external wound. In closed injuries the chances are very high that it is a lesion in continuity (axonotmesis) and that full recovery will occur even though there may be initially reaction of degeneration in the muscles. In contrast open wounds associated with a degenerative nerve lesion nearly always require surgical repair.

Closed injuries. An expectant line of treatment is pursued. The limb is kept in as good trim as possible by frequent passive movement and if necessary splintage and the paralysed muscles may be stimulated by galvanic current.

FIG. 7. Neurapraxia.

The only indications for operative intervention is when recovery is overdue, assuming that the rate of functional recovery is about one inch a month. Thus in a fracture of the humerus associated with a radial nerve palsy the distance from the fracture site to the brachio-radialis muscle is measured. If this distance is three inches then one is justified in waiting three months before considering operative exploration of the nerve.

Open wounds. If an external injury is present and there is evidence of a complete interruption, the wound must be explored and the nerve examined. Immediate primary suture of the nerve should only be considered if the wound is a clean one and if healing by the first intention is reasonably likely. In all other instances it is best to tack the nerve ends together at the primary examination so as to prevent nerve retraction and preserve the correct orientation then when the wound is soundly healed at about three weeks formal exploration is carried out and a secondary nerve repair performed.

The ends of the cut nerves are held together by epineural sutures using fine silk, human hair or steel wire. It is important that the material should be non-absorbable. Capener[3] has devised useful support for paralysed muscles in the form of 'lively splints'. These have the advantage of allowing full extensibility of the paralysed muscles when the active group contract; but at rest both groups are held in the optimum position of rest. For example, when the radial nerve is damaged, a cock-up splint worn for a long period with the fingers extended, leads to loss of extensibility of the extensors of the fingers and the patient cannot make a fist. Unless the paralysed muscles are supported by one of Capener's 'lively splints' it is important to remove any other

type of splint at least once each day for a short period to put the paralysed muscles through a full range of passive movements. This will ensure that the muscles will not lose full extensibility. In addition interrupted galvanism when possible should be carried out. In this way interstitial fibrosis and the other harmful changes in the muscle are delayed.

Prognosis

(a) *Neurotmesis:* Recovery can never be perfect after a nerve suture simply because exact coaptation of appropriate Schwann tubes is impossible. However, protective sensibility should be restored to the previously anaesthetic zone and good recovery of muscle power depending of course on the site of the injury. In general the prognosis in children is rather better than in adults.

(b) *Axonotmesis:* Recovery in these lesions is virtually perfect—provided that the limb and muscles are kept in as good trim as possible by adequate physiotherapy.

(c) *Neurapraxia:* Recovery is perfect and rapid.

REFERENCES

1. Wynn-Parry, C. B. (1961.) *Journal of Bone & Joint Surgery*. Vol. 438, p. 222.
2. Seddon H. J. (1959). War injuries of peripheral nerves, *Brit. Jl. of Surgery*, War Surgery Supplement, No. 2, p. 325.
3. Capener, N. (1949). *Postgraduate Medical Journal*, 25/21.

INJURIES TO THE ARTERIES

SOL COHEN
F.R.C.S.

THE LAST decade saw particularly striking advances in our knowledge and techniques of vascular surgery. With it came a militant aggressive attitude in the management of the traumatic vascular lesion, especially in the young. Prompt repair and restoration of vessel continuity became the endeavour, except in the very ill. Not all surgeons will have served the necessary apprenticeship, or are technically equipped; but straightforward suture repair is well within their ken. Vascular damage is rare in sport; but with motor-cycle and

FIG. 1. Improperly placed haemostat inflicting unnecessary damage to collateral and main vessels.

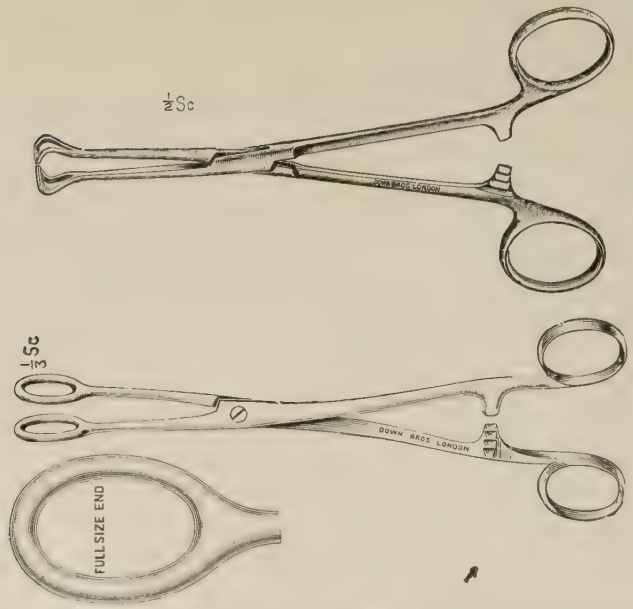

FIG. 2. *Above:* Babcock's forceps. *Below:* Sponge-holding forceps

car racing the doctor in attendance must be prepared for vascular lesions amongst spectators as well as competitors.

Guided athletic exercises have an important role in the management both of arterial and venous disorders. Medical teaching now appreciates and preaches the needs for balanced, guided exercise to step up pulmonary ventilation and assist collateral circulation development in the coronary insufficient or claudicant case. A knowledge of the recent advances in the management of the arterial block lesions of the ageing groups is thus a sine qua non. In vein thrombotic lesions, the vein clot almost invariably absorbs completely leaving valveless veins, and only later (often five to ten years), follow the complications such as recurrent thrombophlebitis, fat necrosis and fibrosis, and finally ulceration. The emphasis is thus on muscle activity to build up the 'peripheral venous pump'. An attitude of invalidism—'take it easy'— is vigorously denounced. These patients need to be taught to walk, to run, to live to the full. They may play tennis, hunt, dance, and some men, suitably protected, do in fact play rugby. Their varicose veins are no longer tolerated, for they do not function as 'bypasses' but add venous hypertension load to the limb. The modern vein stripping procedure gives first-class results, and of course leaves the main draining vein trunk untouched.

Lastly, there is now an answer to the renal failure ('tubular necrosis') that frequently follows extensive trauma, often associated with vascular injuries. With any oliguria the protein and fluid, especially electrolyte, intake needs to be drastically reduced, and renal dialysis may be essential. Transfer, often preferably by air, to one of the excellent specialised Renal Units now available, should be early planned and organised.

VASCULAR TRAUMA

First Aid

To avoid inflicting further injury in the first-aid efforts is an elemental self-evident truth. The particular temptation is blind grasping with haemostat, in the blood-filled wound, for the bleeding vessel and thus adding damage to satellite veins and nerves (Fig. 1). Commonly the bleeding is venous, and often a collateral arterial branch rather than the main vessel. Local gauze packing compression, plus firm elastoplast strapping almost invariably succeeds. In the axilla, or the groin, this may not be possible, and the index finger in the wound is then the best temporary haemostat. With bleeding temporarily controlled, local anaesthesia is injected, and the wound extended, preferably with scissors cut to enable more adequate viewing of the bleeding point. A sponge-holding forceps or a Babcock's forceps (Fig. 2) is ideal in such

emergency for both vein and artery bleeding, and will not add further vessel damage. The use of the tourniquet is almost invariably condemned, but despite its hazards it has a thoughtful temporary place. If the bleeding site is suitable a pressure cuff, supported by a cotton bandage, is to be preferred; soft rubber tubing is an alternative, but must be applied with sufficient force to arrest bleeding. The solid Samway tourniquet is to be condemned as brutal. For the actual vessel repair a proximal tourniquet should almost invariably be at hand.

The *First-Aid Bag* should contain six tubes of 5/0 arterial silk on fine round-bodied curved needles: two each Potts and Blalock's clamps (Fig. 4); two sponge-holding and two Babcock's forceps, a good length of soft 0.5 cm. tubing; surgical haemostatic gauze; two 5 ml. bottles of Heparin (5000 units/cc) and 10 ml. 'Polybrene' for reversing its action. Several ampoules of 2·5 per cent Papaverine.

Blood loss replacement is given priority. The thigh of the fractured femur may contain a litre or more of blood; bruised tissues hold vast quantities of blood and serum. Sufficient blood should be given to maintain the systolic pressure over 100 mms. Hg., and the pulse below 100/min; the urine output should be watched and average 30-50 cc's/hour. The external jugular vein is a good guide to circulatory overloading. With car racing injuries there may be cardiac contusion from the steering-wheel, giving rise to typical myocardial infarction pain, and irregularities. Electro-cardiography check is important.

Vessel repair

Proximal control is the first step, and the skin incision should be extended

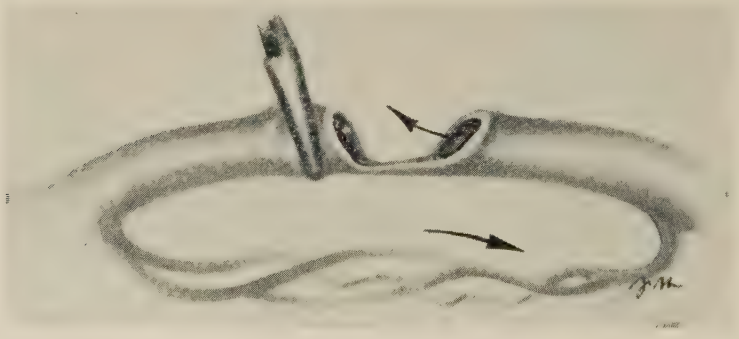

FIG. 3. The Henle-Coenen phenomenon—free bleeding from the distal vessel end; good evidence of a well-developing collateral circulation and absence of clot.

in the line of the vessel length. Umbilical tape (preferably paraffined) or capillary tubing is then passed proximal and distal to the injury lesion and the damage extent inspected. The bruised adventitia should be thoroughly cleaned, for often it masks additional injuries, and in any event is inclined to entangle in the stitches, be drawn into the vessel lumen, and encourage thrombosis. Back bleeding from the distal arterial lumen—the Henle-Coenen phenomenon (Fig. 3) is a pleasing finding, and indicates a good collateral flow. (This sign is not absolutely reliable for the collateral pressure flow may yet be inadequate to overcome distal vessel resistance.) De Bakey (Potts) clamps are now applied at 1 cm. distance, on either side, above and below the vessel laceration (Fig. 4).

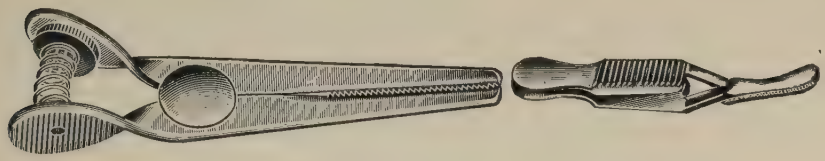

FIG. 4. *Left:* Potts clamp. *Right:* Blalock's cross action haemostat.

The clean cut vessel wound, especially after glass or sharp instrument, needs no debridement. Broad trauma, such as the direct blow and especially the high-speed missile injury, leads to extensive vessel damage, which

FIG. 5. Vessel anastomosis.

commonly has to be excised and the vessel gap bridged. Prior to starting any suturing it is important to be sure that the distal vessel is free of clot and local suction and gentle proximal 'milking' may be necessary. Due to spasm the mouth of the distal vessel often is small and contracted, and may require gentle forceps stretching and local application of 2·5 per cent papaverine.

For the limb vessel repair eversion stitches are no longer used, and the running stitch is now routine (Fig. 5). In the end-to-end anastomosis the one-layer running stitch with two coaptation sutures only is employed; each stitch is inserted 1 mm. from the wound edge, and the 'bites' are 1 mm. apart. When half to two-thirds of the suture is completed, the clamps are alternately released to clear any local clots, and the area washed with 1 per cent Heparin solution. After completing the anastomosis, in order to avoid immediate excess strain on the tightening suture line, the distal clamp is released first. As the suture tightens any suture line bleeding commonly stops. Three to four minutes wait is essential, and the anastomosis is during this time covered with dry or 'Surgicel' gauze. An additional odd stitch or two may be necessary. For smaller vessels the wearing of a binocular loupe (Fig. 6) for more accurate stitch placing is helpful. With a successful suture distal pulsation is immediately achieved. Its failure often means distal clot, and retrograde flushing via the posterior tibial artery at the ankle may become necessary.

FIG. 6. Binocular loupe.

FIG. 7. Retrograde flush.

(Fig. 7.) Post-operative anticoagulant therapy is rarely employed, but may be necessary for smaller vessels; subsequent wound drainage is then essential.

TRAUMATIC VESSEL DIVISION

This may be partial or complete.

The partial injury. The teaching to excise longitudinal wounds and suture these either transversely or longitudinally has now been abandoned. It is impossible to suture a longitudinal wound transversely as is done with the bowel; even with a small defect this leads to vessel 'knee kinking' and clotting. A narrow longitudinal vessel injury can, of course, be sutured. (Fig. 8.) But a more than minimal rent should be vein patched (Fig. 9); otherwise lumen narrowing and local clotting follow. Such vein patching enlarges the calibre, 'arterialises' and aneurysm formation does not develop. The patch is taken from a split long saphenous vein; the upper extremity and external jugular veins are too thin and hence unsatisfactory. A badly damaged vessel segment is better excised and if the gap is small, a fresh end-to-end anastomosis fashioned.

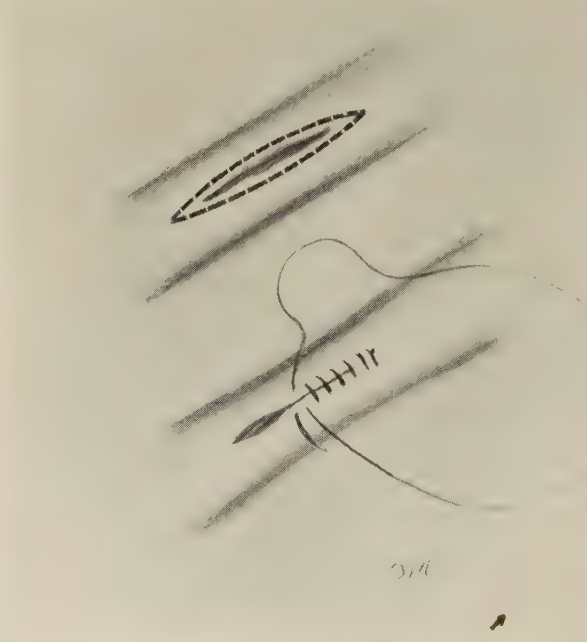

FIG. 8. Suture of narrow longitudinal tear.

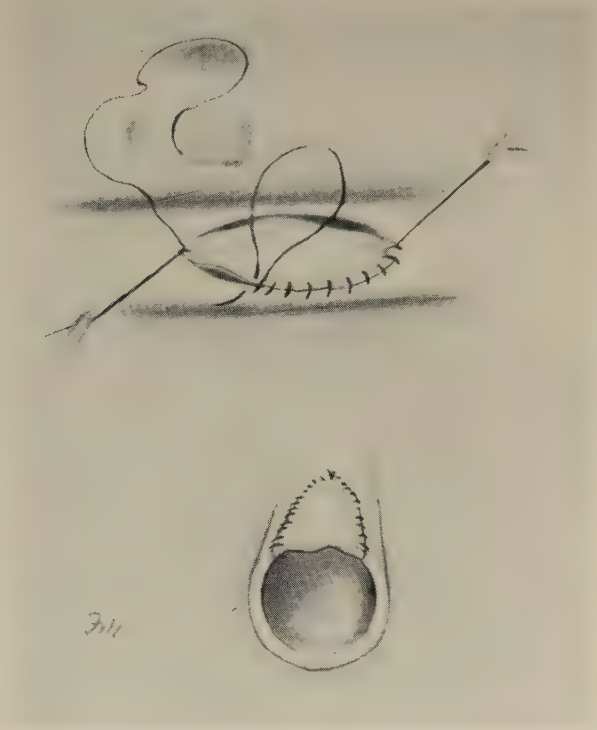

FIG. 9. Vein patchgraft.

Complete division

Due to retraction, often remarkably little local bleeding is found with the completely divided vessel. The bruised ends should be trimmed preferably with razor blade, but scissors are permissible. The vessel should be cleanly dissected for about 10-15 cms and the applied clamps gently approximated to estimate the feasibility of repair, or as to whether grafting is essential.

Grafts: It is now agreed that the autogenous vein graft is to be preferred: cellular viability is assured, and the ultimate results are good, but technically, the procedure is more difficult (Fig. 10). The saphenous vein of the injured side is commonly employed; but if the deep vein has been damaged, the saphenous vein on the opposite leg should be used, and is, of course, reversed. Stored arterial homografts have been found disappointing, almost invariably calcifying, forming aneurysms and thrombosing over the years.

Dacron grafts are a preferable alternative, but are also rather liable to clot in the limbs over the years. They are readily available, easy to handle, and should be used when time is important.

Vessel contusion

This may be a *minor local bruise*, and no surgery required. It does not cause local or distal vessel reflex spasm. The *major contusion* is a serious problem. Frequently, there is extensive intramural bruising, intima disruption and coiling, and the lumen blocked with clot. Local clot removal is almost invariably followed by reclotting. Excision of the contused segment, with preferably vein or Dacron prosthesis grafting, is often essential. Distal thrombus clot is a common finding in these cases, and ankle retrograde flushing may have to be undertaken. The fixation of associated fractures in long bones is important, and the Kuntschner nail is a good method. It is to be remembered too, that with a sharp external blow, there may be no skin damage, yet the vessel intima be severely contused, or disrupted, and blocking flow.

Vessel spasm

Arterial spasm is often unnecessarily invoked as the causa causans of limb ischaemia. Absent pulses and ischaemic signs are more commonly due to lumen clot. Without vessel exploration, it is impossible to differentiate between vessel disruption, thrombosis or spasm.

The loss of distal pulses after femoral, tibial or humeral supracondylar fractures is often due to longitudinal stretching over bone fragments. Occasionally, there is haematoma compression, or actual bone spicule blocking vessel flow. The most effective local spasm relaxing agent is Papaverine (2·5 per cent) on a gauze soaked swab—it takes 15-25 minutes for its full effect. After severe haemorrhage, vessels are often 'irritable', and assume tonic state on handling.

Ligation

Lengthy operations on exsanguinated patients are, of course, irrational: better to lose a limb than a life. When ligature becomes essential, fine silk doubled or trebled is the preferable material. The limb survival then depends on the vessel and the local collateral vessel damage. The popliteal and common femoral arteries are the *dangerous 'critical' vessels;* the brachial, the subclavian and iliac vessels, can commonly be ligated with impunity. Damage of the crutch bifurcation of a main vessel is particularly serious; and injury to both distal limb vessels, e.g. the tibial and peroneal, or the radial and ulnar,

may be followed by gangrene. Return flow from their distal stumps, is a good sign.

A *line of demarcation* commonly develops in three to four days: if confined to the foot or hand, there is very little 'toxic' absorption, and it is safe to wait. With muscle death, as in the leg or thigh, cell potassium release is the particular hazard, and sudden death may follow. Daily serum potassium check and urine output are essential. Insulin (20 units) will reduce the serum potassium level, and exchange ion resin enemas, may be required. If there is any oliguria, progressive rise in blood urea or potassium, or infection, amputation may have to be expedited.

Coincident vein ligation is no longer considered of advantage, and may lead to unnecessary, permanent limb swelling. Sympathectomy or sympathetic blocks for the 'acute' vascular lesion may be harmful, and shunt blood from the main distributing stream. These procedures are reserved till later date,

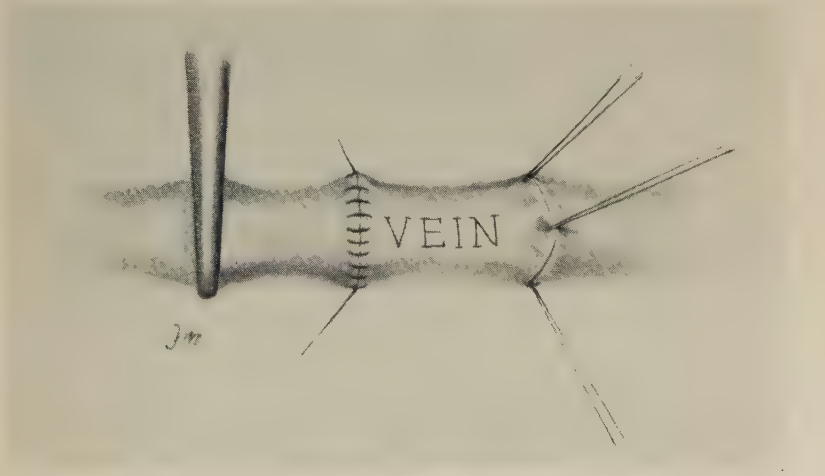

FIG. 10. Vein graft replacing excised arterial segment.

when the collateral circulation is more fully developed. *Vasodilator drugs* are inadvisable, and in view of their wide unselective vaso dilative effect, may indeed shunt blood away from the ischaemic limb.

Venous trauma

Venous bleeding may be copious, and the visulation of the extent of venous damage difficult. It is most effectively controlled by an assistant gradually spreading from the middle of the wound two vertically held swabs on forceps

or tongue spatulae. (Fig. 11.) The large vein rent may be temporarily grasped with Babcock, or swab holding forceps. Small veins are, of course, ligated; large vein tears are easily sutured with 5 × 0 arterial silk. Vein grafts to replace damaged vein segments are inadvisable, for clotting with pulmonary embolism is the hazard.

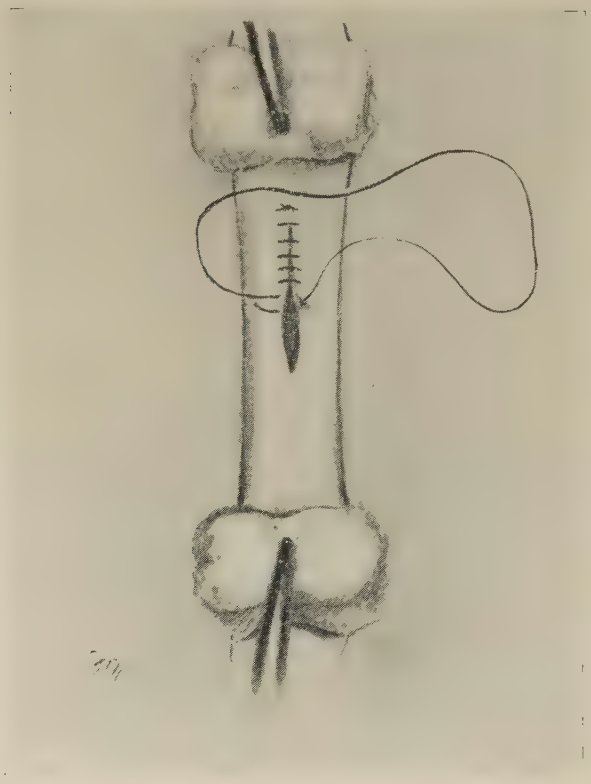

FIG. 11. Control of venous rent bleeding.

Extensive venous trauma may be followed by the dreaded Volkmann lesion. Muscle death ensues in manner akin to that of the complete arterial block lesion, and the microscopical picture is that of muscle replacement fibrosis. Such extensive venous damage is not unusual after motor cycle injuries, and humeral supracondylar fractures. In the swelling limb encased in tight plaster, there is similar vein compression effect, with often in addition

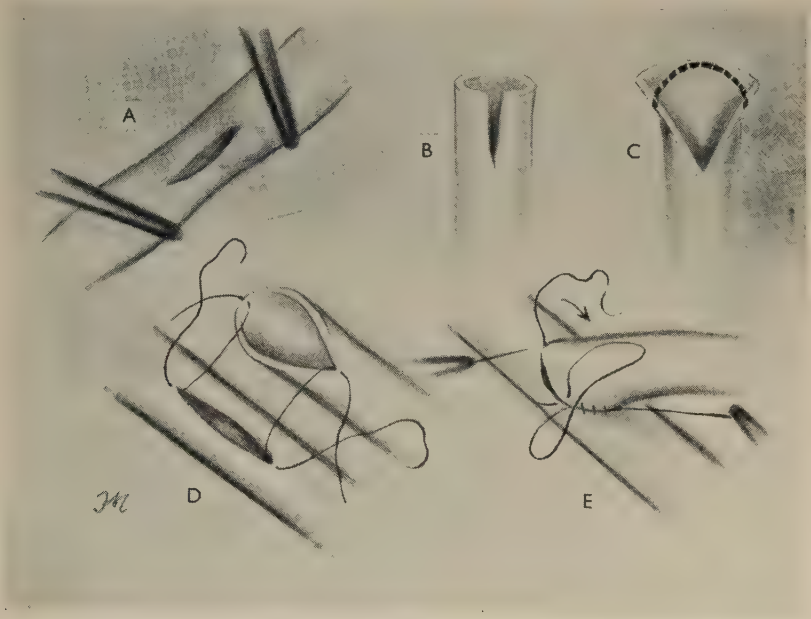

FIG. 12. Technique by-pass graft—required for extensively rent or contused arterial wall.

ischaemic distal neuropathy. The decompression of such a limb is important and early longitudinal muscle *fascia splitting* (formerly condemned), is now practised. It releases the pressure on the residual veins. In doubts as to limb viability, any plasters should be completely removed, rather than merely split.

Ischaemic signs

Loss of muscle power is the most important. It takes 30-40 minutes to develop after the injury, and is associated with distal sensory loss. Inability to move the toes or fingers, or dorsiflex the foot, calls for action. The dying limb commonly feels cold and numb to the patient or may be exquisite agony. The waxy pallor and cyanosis of the ischaemic limb are well known, but it needs to be emphasised that brisk return flow after local nail pressure is no guide to the arterial circulation. This is related to high venous tone, and severe arterial arrest may yet be present. In the doubtful case, arteriography has a place—and is safe.

The ischaemic limb itself should be nursed in a slightly below heart level

position and protected with a cotton wool muff from cold, local heat or cradle injury.

SPECIAL CASES

The *fractured femur* often has associated main vessel injury—either disruption, severe contusion, or impaling by a bone fragment. Owing to associated extensive collateral vessel damage in such injury, death of the limb is certain, unless restitution surgery be undertaken. It is commonly essential with such vascular surgery for the femur to be first fixed by Kuntschner nailing. The rule is—watch the distal pulses and toe movements; if at the end of two hours after injury these have not returned, exploration is essential. The anterior *shoulder dislocation* may tear the axillary artery with loss of distal pulses. Arterial repair is usually feasible. Occasionally, the scapular artery only is torn, and thrombosis extension into the main vessel is found. Falling on the tip of the shoulder without dislocation, has been known to tear in similar manner the axillary artery, and a thin strand of tissue, mistaken for arterial spasm, is found. This arterial segment, commonly 1 cm. long can be resected and resutured. The *dislocated knee-joint* almost invariably tears the popliteal artery, and restorative surgery is absolutely essential. Dacron grafting is the preferable choice.

Fractures of the tibia and fibula : Arteriography studies show that the vessels often kink in remarkable manner around the bone ends. Increasing the limb traction often unkinks them.

Crutch axillary arteritis : The hazards of axillary crutches are well known, but such axillary artery thrombosis is still being encountered, and the remedy is obvious.

Time lag of repair : Tissues vary in their ability to withstand complete arterial arrest. Skin and nerves will recover even after 24 hours, but muscle can withstand only some six hours ischaemia—under cold conditions, possibly up to ten hours. To restore the circulation flow after six hours, may be followed by sudden death of the patient, from potassium and other products of the dead muscles. I have seen this happen on a number of occasions, and this is an important consideration in late vessel repair.

Pulsating haematoma : With partial vessel injuries and small wounds, a localised pulsating haematoma, and later a 'false' aneurysm develops. The arterial haematoma pulsates, and within hours, a 'localised' systolic bruit is heard. There may be, in addition, direct communication with the vein, and an arterio-venous fistula form. A continuous bruit (machinery) 'widely propagated' and with systolic intensification is then heard. Both types of aneurysm lesions are better left unoperated for some six to eight weeks. In

both, distal pulses continue, and can be checked by oscillometry. Daily circumference measurements of the arterial haematoma are important.

In boxing superficial temporal artery aneurysms, and in basketball digital arterial aneurysms, have been encountered. Such lesions have also occasionally followed partial genicular artery injury in knee-joint surgery; and common iliac artery fistula has been a serious complication of lumbo-sacral disc surgery, in quite a number of recorded cases.

Sudden death in athletes: This has occurred occasionally after severe exercise. There are three particular causes:—(a) coarctation of the aorta. Hypertension commonly accompanies this and athletes undertaking weight-lifting should invariably have blood pressure check. (b) medial necrosis of the aorta as in Marfan's disease. This is commonly a familial disease and the patients are usually of the tall, gangling type ('Basketball player'), with long arms. (c) thoracic aortic aneurysm. Occasionally, after automobile accidents, an aneurysm at the arch of the aorta develops. This is secondary to the sudden acceleration destress. Chest injuries should have repeated X-ray check. These aneurysm lesions are slow growing, and survival periods of 15-20 years have been reported.

Lastly, as already indicated, myocardical contusion should always be borne in mind after the severe motor car or cycle spill.

EFFORT THROMBOSIS

Strenuous athletic activity may on exceptional occasion be followed by *major vessel thrombosis*. Perhaps the most dramatic is the story of Dr. Rivers Pollock who in 1844 won the Oxford/Cambridge 120 yards hurdle race for Cambridge University in what was then the record time of 16 secs. During the entire race he held his breath.

'Immediately after the race he complained of severe lumbar spinal region pain and was put to bed, where he remained for six months. Oedema of the legs and, to a lesser extent, of the abdomen and scrotum supervened at once . . . . albuminuria appeared directly and persisted throughout life. Dr Pollack died some twenty-five years later from tonsillitis and septicaemia. At autopsy the inferior vena cava was a flat impervious ribbon, and the common and external iliac veins were similarly obliterated. Kidney venous return was via capsular veins into the lumbar veins, and then through the enlarged azygos vessels. Dr Shattock was of the opinion, that following breath-holding there was localised rupture of the intima and then vein wall dissection in manner akin to that of the dissecting aneurysm of the aorta'

(Pollack *et al.*, 1956).

Iliac or femoral vein thrombosis has been an occasional complication of in particular strenuous cycling efforts. Improper breathing of the competitor during exercise is a likely factor. Straining with closed glottis and contracted abdominal muscles carries out 'Valsalva's manoeuvre', which impedes venous return to the heart and this may lead to venous intima damage and thrombosis.

Subclavian vein thrombosis following unaccustomed effort is a well-known clinical entity. It has been provoked by handling a 'high-spirited' horse, rowing, tennis, and lifting heavy objects (Kleinsasser, 1949). External jugular vein thrombosis has been seen following strenuous effort.

Immediate Heparinisation (12,500–15,000 units) is the answer, and is to be preferred to the oft advised stellate or lumbar sympathetic blocks. These cases should be thoroughly investigated as to any thrombotic accelerating factors, such as blood dyscrasia (e.g. leukaemia) or any occult malignant lesion, especially the lung. Hyperuricaemia (with no gouty attacks) has been a finding in a number of cases. Indeed such subclavian thrombosis is often described as 'Paget's gouty phlebitis'.

The *Anterior Tibial syndrome:* ischaemic necrosis of the extensor leg muscles and anterior tibial nerve is of particular importance to athletes, and has followed playing football, running, or jumping on pavements. It has never been described in the professional footballer or the trained athlete, and has only occurred in those unaccustomed to exercise. The mechanism is as yet uncertain; but following muscle fibre tear, haemorrhage occludes the muscle branches, and vasa nervorum, and a typical muscle Volkmann lesion follows. There is exquisite pain over the extensor compartment of the leg, the skin of which becomes glossy and tense. Secondary to the associated nerve compression, there is anaesthesia of the dorsum of the foot, and the area between the big and second toes. Early longitudinal decompression incision of the fascia (within six hours) may save such muscle death.

Arterial thrombosis seldom follows effort but exceptional cases do occur. Subclavian artery thrombosis has been described following the throwing of a cricket ball by a thirteen year old girl (Jepson). Cervical ribs, thoracic inlet abnormalities and Collagen diseases should be searched for in such unusual cases. Digital artery thrombosis I have seen follow the 'stubbed' shot at golf. Walking over ploughed fields, riding, digging in the garden have occasionally been followed by an acute popliteal or femoral arterial block lesion, but invariably there has been underlying extensive arteriosclerotic disease.

Haemangiomata: in young servicemen, with the initiation of vigorous training, it is not unusual to find the effloresence of the apparently quiet haemangioma, and this may be mislabelled malignant. The increased local skin and muscle blood supply after exercise feeds the haemangioma blood flow and it

enlarges. On the scalp a minor angioma may develop into the full cirsoid aneurysm lesion.

VENOUS DISORDERS

Varicose veins

The long distance runner, in particular, often has a markedly prominent superficial venous system. These veins are not varicose, not incompetent, and 'phlebectasia' is their label. Their ablation is a mistake, and surgery may be followed by deep vein thrombotic complication. Vessel cramps at night in bed or on the field are not due to such phlebectatic veins, and more commonly are related to overtraining and chloride loss by oversweating. Many of these athletes should be instructed to change their jerseys when soaked, and take extra Bovril or Oxo; the salt tablets are commonly rejected as unpalatable. True varicose veins, which are often familial, do however require surgery, and with the modern vein stripping procedure first-class lasting result can be promised. Injection therapy for even the most puny varicosis is to be absolutely condemned; for the sclerosant flows into the deep veins and may add deep vein valve damage.

The deep vein thrombotic lesion

With a better understanding of the venous haemodynamics treatment of these cases has become rewarding. The essential endeavour is to build up muscle tone (the peripheral venous heart) by active exercise and thus compensate for valve incompetence. The need for active exercise must constantly be preached. 'Take it easy' is grim fatal advice of the past. They must reduce weight, sleep with eight inch bed blocks, and elevate the leg at rest; mild chlorothiazide therapy may be necessary to initiate leg oedema reduction. Elastic stockings should be worn. These should be of below-knee length, made of Lastex yarn, and changed every three to four months. The above-knee stocking rucks, slides and is inefficient. For social occasions the stocking may be discarded, or the American type of nylon stocking worn. It is important to avoid fostering an attitude of invalidism. With ankle induration, local thrombophlebitis and fat necrosis, threatened ulceration, careful palpation for ankle 'blow-out' feeder veins is important. Their eradication by surgery—Cockett's procedure—gives first-class lasting results.

Heparin is the only satisfactory anticoagulant agent for the acute thrombotic lesion and should be continued for seven days. The Swedish dosage schedule is to be recommended, i.e. 15,000 units: 10,000 units: 10,000 units: 15,000 units: at six-hour intervals, via an intravenous Gordh needle. Spon-

taneous haemorrhage does not occur and clotting-time checks are unnecessary. Heparinisation is dangerous for the fracture in tight plaster. Oral anticoagulants (Dindevan) start at the end of a week, and are used for prophylactic purpose (the unaffected leg), rather than the established thrombotic lesion.

Superficial thrombophlebitis

This may follow prolonged plane flight, a severe cough, or pneumonic infection; occasionally there are unexplained, apparently viral, minor phlebitic epidemics. In older patients, thrombophlebitis out-of-the-blue often has sinister significance, and may be the first evidence of an unsuspected growth. Superficial thrombophlebitis is painful and disabling, but the lumen clot is tarry or fixed and patients may be reassured as to any pulmonary embolism risk. Rest in bed is, however, dangerous, for clotting is then liable to spread via the anastomotic vein to the deep vein tree. The area of phlebitis should be firmly strapped and full ambulation encouraged. Heparin (15,000 units twice daily for 4/5 days) often gives dramatic spectacular relief of pain. Penicillin therapy, so commonly used has no rationale or sanction.

ARTERIAL DISORDERS

Intermittent claudication of the calf is the common presenting complaint, but this may start in the buttock or thigh, and frequently simulates the hip arthritic lesion. The essential features in the diagnosis are: the fixed walking distance for its onset, its relief is under two minutes on halting, and *without* the need for change of posture of the leg. These patients will, of course, be studied to place the site of the block by oscillometry and arteriography; and for evidence of any thrombotic factors such as hyperglycaemia, hypercholesterolaemia, etc. For the 'above-inguinal' ligament block, surgery has a good deal to offer. 'Decoring' or reaming out of the aorta or iliac vessels, are now safe procedures, and local dacron graft results are pleasing. 'Below-the-inguinal' ligament grafts have proved disappointing and are rarely to be sanctioned for the pure claudicant case, with no distal nutritional anxiety. Within two years the majority of such grafts, especially cloth or stored homografts, clot.

Sympathectomy is reserved for the cold foot, ischaemic resting limb pain or threatened gangrene. Tendo Achilles section and calf neurectomy have proved disappointing; the subsequent slapping limp is resented, and often followed by knee or hip arthritis.

Most patients can be taught to dodge their claudication pain, by adopting

a slower walking pace. They need to be told also that the onset of pain need not be feared, and will not provoke further thrombotic spread or gangrene; and gentle walking is the best of all measures for developing the collateral vessel blood flow. Patients often enquire as to whether they should abandon their smoking habit. *Cigarette smoking* should be entirely abandoned, but the odd cigar and pipe are, at the present state of our knowledge, permissible. Cigarette smoking reduces coronary flow, and it is the coronary vessels that form the long-term concern, even in the peripheral arteriosclerotic.

Thromboangiitis obliterans (Buerger's disease) has recently been described as but 'arteriosclerosis developing in the young'. Opinion in this country, and certainly the Mayo Clinic, continue to regard this as a definite distinct clinical entity—an 'allergic vasculitis', probably a variant of polyarteritis nodosa. Smoking must be entirely abandoned; if patients can be persuaded to do this the march of the disease is commonly halted.

Younger patients, even athletes, are often concerned about their fingers becoming pale or cyanosed in the cold weather, and the diagnostic label of *Raynaud's phenomenon* or disease alarms them. This increased arterial sensitivity to cold is often familial, a mere nuisance and patients can be firmly reassured that total finger or hand gangrene will not follow. They are often made worse by large doses of vasodilators. They should of course be investigated to exclude any underlying Collagen disease or hypothyroidism. A few are helped by 'cervical' sympathectomy. Hyperidrosis, i.e. pouring sweat from both hands or feet is an occasional complaint of athletes. Sympathectomy abolishes for all time in dramatic fashion such sweating.

RENAL COMPLICATIONS

With, in particular, motor car/cycle racing injuries, there is need to be alert to possible renal failure complications. If the patient be unconscious or semiconscious, and there be any anxieties re the urine output, a Gibbon's catheter should be inserted. If the urine output is under 400 ml. daily, this is classed as oliguria. The great danger is to assume that the patient is dehydrated and push the intravenous or oral fluids. Unable to excrete the fluid, the patient verily drowns. The answer is forthwith to reduce drastically fluid intake to 400–500 ml. daily, plus any sensible fluid loss, e.g. urine output. Anabolic steroids—50 mg. Norethandrolone daily—are given to reduce protein breakdown. A protein-free diet is essential and 100 g. glucose daily is now routine. No antibiotic drugs other than penicillin are permissible, and for sedation

Phenergan or chlorpromazine are used. Transfer of the patient to a renal unit is to be planned.

TRANSPORT

Patients with associated fractured femur, pelvis or spine travel badly by ambulance, but helicopter or fixed wing flights can now readily be arranged. Flights by B.E.A. or Service helicopter average £100 per flying hour; by Dakota about £80; and by 'Rapide' £20 (Skone and Mills 1960). The National Health Service has been willing to provide the cost of such transport.

To transport the patient to and from the take-off and landing sites in greater comfort, a plaster bed is valuable. Pilots should fly preferably not higher than 2000-3000 feet, especially if the patient's haemoglobin level is low, and if there be any associated chest injuries. Despite the noise, such helicopter transport is comfortable, the landing gentle, and often life-saving when the patient is handed over to, say, a well-organised Renal Unit. The oliguria of the acute renal failure commonly lasts two to three or more weeks, and during this time they are subjected to intense 'starvation'. Three or more artificial kidney washes (dialyses) are required and this will depend on the blood urea rising rate. This 'renal tubular necrosis' appears to be completely reversible, and I have known them subsequently absolutely fit, play tennis and even undertake deep sea skin diving.

REFERENCES

Kleinasser, L. J. (1949). Arch, Surg. 59, 258.

Pollak, V. E., Kart, R. M., Pirani, C. L., Shafter, H. A. and Muchrcke, R. C. (1956). *Am. J. Med.* 21 (No. 4), 496.

Blagg, C. R., Parsons, F. M. and Young, C. A. (1962.) *Lancet*, 1, 608.

Skone, J. F. and Mills, J. (1960). *Lancet*, 2, 363.

5

UPPER LIMBS

THE NEXT three chapters deal in some detail with specific injuries, stressing especially the importance of immediate treatment so as to prevent certain avoidable complications. They are not intended as a treatise for the orthopaedic specialist, but rather as a guide to the general practitioner, especially the school doctor, the medical officer in the armed forces and all those doctors associated with athletic organizations.

It is unfortunately true that the medical profession as a whole does not yet realize the importance of treating contusions and bruising of the tissues immediately and actively. So often the patient is told to rest and nothing further is done. We have already stressed that contusions and bruising of tissues often lead to painful thickenings and adhesions, muscle atonia and wasting. These complications can be avoided if, at the same time as the injured parts are protected from overstrain, they are also exercised without strain, and given injections locally and physiotherapy. Many thousands of patients have now been treated by active methods, and we propose to describe the methods which we have found to give the best results and the earliest return to fitness in each type of injury.

When part of the limb is injured, it is essential, let us repeat, to think of the limb as a whole. So often a trivial injury in one area will affect other areas. A finger injury, for instance, if left untreated, may produce strains at higher levels in the arm, so that, even the shoulder girdles and cervical vertebral joint muscles may become involved. Similarly, faulty foot posture may start progressive strains which eventually cause pain in the buttocks and the back.

THE PHALANGES AND HAND

The fascial compartments of the fingers and hand are so thick in many places that traumatic effusions have great difficulty in spreading or becoming absorbed. The hand, because of its dependant position, is not an area from which blood and lymph are readily absorbed. Distension of the tissues of the hand and fingers by traumatic effusion may have an unfortunate and sometimes disastrous effect on the blood supply. In finger-joint sprains, for ex-

ample, when a contusion or sprain occurs, the whole circulation of the part is severely disturbed. Unless the correct balance between rest and movement is established, traumatic effusion remains unabsorbed and tends to form adhesions and thickening, giving rise to stiffness and limitation of movement.

Sprains of the inter-phalangeal joints and metacarpo-phalangeal joints

The joint requires protection and support. A removable plaster slab or suitable malleable duraluminium splint covered with dalzifoam is adequate. The new tubofoam splint is ideal as it can be taken off easily and the part given home treatment as well as physiotherapy. If organized physiotherapy cannot be given more than once a day the patient should himself carry out home treatment several times each day. Wax baths and anodal galvanism are particularly good for this type of injury, which ought to be treated as early as possible. Even large effusions can be made to absorb within two or three days; but if left will give rise to a chronic stiff joint. Injections of procaine or xylocaine with hydrocortisone and hyaluronidase may be necessary and help to disperse and absorb the effusion, they may have to be repeated more than once. A useful tip is to strap the injured finger to its neighbour, which serves as a splint and also induces some active use of the injured finger.

These injuries may be complicated by chip fractures of either phalanx and they can usually be treated as severe sprains. The full range of movement must become re-established before the finger can function fully, and to effect this, gentle manipulative movements are started within the first few days. These are carried out so gently that there is no undue reaction. If there is not daily improvement the treatment is too vigorous.

An example will serve to show the course of these injuries when they are properly treated. A county wicket-keeper sustained a chip fracture of the terminal phalanx of the left ring finger, with gross swelling of the terminal inter-phalangeal joint. The joint was stable. Treatment given was:- protection and support by a removable padded plaster of Paris finger stall; wax baths; massage and faradism twice daily for three days; and regular home treatment. At the end of three days all swelling had subsided and there was full movement, but the terminal inter-phalangeal joint was still tender. He was advised to leave the finger in the plaster support without removing it for a further three days while he carried out strong isometric movements of the terminal inter-phalangeal joint against the resistance of the plaster. At the end of six days the inter-phalangeal joint was normal, and he started to play cricket, which included wicket-keeping. On the eighth day after injury

in his first match, he caught three players in the first innings and stumped four in the second; which suggested that the finger had recovered full function.

Complicated cases

Stiff interphalangeal joints: a simple sprain of an interphalangeal joint may lead to a chronically thickened, stiff and painful joint which swells after exercise. They may be the result either of prolonged immobilization or of insufficient immobilization. If excessive movements are permitted in the early stages after injury, so that the joint becomes inflamed and tender, it may not recover normal function until given appropriate rest.

The patient must be warned that such joints cannot be made symptom free in a short time. They may need two or three months' regular treatment before they are normal.

The patient should be given physiotherapy, daily if possible, but at least three times weekly. It should consist of wax baths, massage, gentle manipulation and faradic contractions. The patient must also constantly carry out remedial exercises and massage himself at least twice daily. The joint almost always passes through a painful phase owing to the breaking down of the periarticular thickening, and the patient must be warned and reassured about this. Gentle regular manipulations are far better than a forced one under an anaesthetic, to which the reaction is usually excessive, and the joint stiffens again before the reaction can be cleared up. It is well to give an adjustable duraluminium splint to rest the finger when the patient is in bed.

In the established stiff painful interphalangeal joint recovery by conservative methods is often so slow and unsatisfactory that arthroplasty may be indicated. Recovery following operation is again notoriously slow but if the patient perseveres with graduated active use, combined with frequent physiotherapy, a good result can often be obtained even if it takes several months.

A chronically thickened stiff, painful metacarpo-phalangeal joint often occurs in boxers and a decision has to be made between capsulotomy and conservative treatment. If the latter is tried it must be concentrated and must consist of weekly injections of xylocaine and hydrocortisone, home treatment three times daily, physiotherapy at least once a day and return to boxing activities slowly, the joint being well supported and protected. Usually in four to six weeks there is so much improvement that the need for operation disappears.

Example: A well-known boxer suffered from chronic thickening of the capsule of the metacarpo-phalangeal joint of the left little finger. He complained that the joint was extremely painful, when he punched with the left

hand. He was given intensive treatment twice daily for two months with one injection of xylocaine hydrocortisone each week and at the same time he carried out home treatment conscientiously. Apart from the home treatment he had about eighty hours of intensive physiotherapy. He was made completely symptom free, and it is doubtful whether operation would have produced a quicker result and allowed him to return to competitive professional boxing so quickly.

Another case, however, illustrates the need for operation. A county wicket-keeper sustained such a blow on the thumb metacarpo-phalangeal joint of the left hand that the capsule on the radial side was ruptured. He was first examined some months after the original injury when the joint was thickened and tender and was markedly unstable. There had been another recent direct injury which had aggravated the symptoms. The player himself felt that he would never be able to keep wicket again due to the instability of the joint. At operation the capsule of the lateral side of the joint was found loose and fibrotic, and there had obviously been a tear of the lateral ligament. The region of the rent was reinforced by a fascial graft from the tensor fascia lata and the player returned to county cricket in six weeks.

Another example was a middle-aged athlete aged 56 who had sustained a blow on the joint of the index finger of the right hand which precipitated arthritis of the metacarpo-phalangeal joint. The joint was thickened, stiff and painful. Movements in every direction were practically nil and the flexor tendon was firmly adherent to the front of the joint capsule. A preliminary course of one injection of hydrocortisone and home treatment for one month while the patient was awaiting a hospital bed improved the condition considerably but since it seemed that the arthritic condition would need continual treatment, operation was carried out. The head of the metacarpal was removed and a fascial graft was placed surrounding the cut end of the neck and active movements commenced immediately in a plaster cast. The stitches were removed in a fortnight and the joint was protected and supported in a removable plaster cast. Home treatment continued and the support and protection was gradually removed. When seen ten weeks after the operation the patient was carrying out full duties as a gardener with a painless fully mobile new joint.

Button-holing of the extensor expansion: An ugly deformed proximal interphalangeal joint can result from the button-holing of the central slip of the extensor expansion over the dorsal aspect of this interphalangeal joint. There is a subluxation at the proximal interphalangeal joint of the head of the proximal phalanx backwards on the middle phalanx. This type of injury requires immediate operation.

Mallet finger

The attachment of the extensor tendon to the proximal end of the dorsal aspect of the terminal phalanx may be ruptured or torn away with a flake of bone. The treatment of this condition is undergoing a definite change. Previously it was considered essential to immobilize both the proximal and distal interphalangeal joints in a plaster of Paris cast for eight weeks. The cast is difficult to apply because the distal joint must be hyperextended and the proximal interphalangeal joint flexed. If this method is adopted the patient should carry out isometric movements against the resistance of the plaster and if possible have surging faradism to the flexor, extensor and intrinsic muscles at least twice weekly. The finger must be held immobilized for eight weeks continuously to be certain of success. Other methods, however, seem to give equally good results: the distal interphalangeal joint may be immobilized by means of two or three pieces of Kirchner wire placed across the joint in the soft tissues at different angles; or by strapping, plaster, or strip dalzifoam; or the patient may be fitted with a plaster of Paris cast which he can take off for active home work and physiotherapy. These latter methods can be employed when there are other injuries, such as damage to the proximal interphalangeal or metacarpo-phalangeal joints. For example, an international wicket-keeper sustained a mallet finger of the little finger of the left hand, but at the same time suffered a chip fracture of the proximal end of the middle phalanx, involving the proximal interphalangeal joint. He was treated by a removable plaster cast which gave support and protection to both interphalangeal joints, and he had active treatment including physiotherapy and home treatment. Luckily, the accident happened at the end of the season, and by the next spring he had made an excellent recovery, the only residual symptom being slight limitation of hyperextension of the distal interphalangeal joint.

Untreated cases often become relatively symptom free if they are given a removable splint and active treatment. Occasionally it may be necessary to perform arthrodesis of the terminal interphalangeal joint to get rid of persistent pain and stiffness.

Trigger or snapping finger

This condition, which is tenovaginitis of the flexor tendon, may follow a direct blow on the tendon sheath, but is more common after sustained movement in which there is a twisting or rotation strain on the finger. Any finger can be involved but in cricket or baseball, it is usually the index or middle fingers—the 'spinning fingers'—which are most affected. The condition may also follow sprain of any of the finger joints, particularly the metacarpophalangeal joint.

There is inflammation of the tendon and its sheath, and in the established case, the tendon becomes enlarged and thickened just distal to a constriction in the sheath with the result that, when the tendon is flexed the thickening snaps through the constriction causing considerable discomfort. When the finger is straightened the tendon snaps back again. The common point of constriction is over the palmar aspect of the metacarpo-phalangeal joint, which is approximately half way between the first crease of the palm and the crease at the base of the finger. To the palpating finger it feels like a small button. If the condition has been present for some time, surgical removal of the portion of the sheath with the constriction is the quickest method of establishing a cure. It may be the only method.

Conservative treatment in the form of physiotherapy and home treatment, combined with weekly injections of xylocaine and hydrocortisone, may produce a cure, especially if treatment is continued for six to eight weeks. About the third week the thickening of the tendon sheath begins to break up and the area of tenderness increases with exacerbation of symptoms. Symptoms tend to recur after conservative treatment because the thickening of the tendon and the constriction of the sheath may never be entirely removed.

If the patient has been treated by operation, he usually needs a period of home treatment and physiotherapy, particularly when inflammation is widespread, involving not only the flexor tendon but the lumbricals, interossei and the capsule of the metacarpo-phalangeal joint as well.

Contusion sprains of the dorsum of the hand

Thickening and swelling of the structures of the hand and fingers due to organization of haematoma may occur between the metacarpal heads and, in certain cases, in relation to the extensor tendon sheaths. In the latter case the painful oedema described by Secretan may be established. Often all forms of treatment for this condition fail to establish a cure, even with local surgery or stellate ganglionectomy.

These are usually produced by a direct blow. Vessels are ruptured, and there may be a large extravasation of blood into the soft tissues. If the patient is seen soon after the accident, the extravasation can be helped to absorb by immediate injection of 1000 units of hyaluronidase given with 5 ml. of 2 per cent procaine, a firm pressure bandage, massage and faradism. If the swelling is under much tension, it is best to aspirate the fluid or express it through small incisions. There is usually no harm in waiting until the following day to see the effect of the injection and physical treatment. The following case illustrates the efficacy of this treatment; a famous cricketer sustained a severe blow on the hand in a test match and was told by his doctor that he could not play for

three weeks. However, he was given an injection of xylocaine and hyaluronidase with physiotherapy. Three days later he was so fit that he took five wickets for 29, and finished by scoring 48 not out.

The severe case

A severe blow, producing extensive contusion of the hand and much haematoma formation, may be associated with other injuries such as fractures of the metacarpals. The condition requires to be taken very seriously and treatment must be carefully supervised until the hand is restored to normal. In a severe injury this may take six to eight weeks. Treatment should include:—

Protection and support—a padded removable plaster is best.
Postural drainage.
Physiotherapy—effleurage and faradism, galvanic or sinusoidal baths.

If there is a large localized haematoma, decompression of the area by incision to relieve tension is often indicated. If the swelling occupies a large area, make multiple small incisions through the deep fascia and then soak the whole hand in cold weak Dettol solution. Faradism and the application of ice packs or cold compresses may also help. Cold is better than heat: in some cases a course of small applications of deep X-ray is indicated.

Contusions in the dorsum of the hand can involve the extensor tendons and cause chronic tenovaginitis, with the formation of tender thickenings. These are extremely resistant to physical treatment when well-established, and treatment may be needed daily for many weeks. Sometimes one can get a quicker result by operation.

DISLOCATION OF THE SEMILUNAR BONE

All injuries to the wrist joint should be X-rayed in four different planes to exclude a fracture of the scaphoid, dislocation of the semilunar or some injury to any of the other bones of the wrist joint and carpus. Watson Jones[1] has described in detail the various types of fracture dislocation occurring at the wrist and carpal joints. Dislocation of the semilunar bone is the next most common injury to the fractured scaphoid. It can usually be diagnosed from:—

1. The history of injury,
2. A painful swelling on the palmar surface of the wrist with evidence in some cases of pressure on the median nerve,
3. Painful limited movements especially flexor,

4. A positive Murphy's sign i.e. pain over the semilunar on tapping over the head of the middle metacarpal bone with the wrist adducted,

5. X-ray findings.

The semilunar can be observed rotated and displaced forwards in relation to the rest of the carpus.

FIG. 1. Reduction of displaced semilunar bone. The thumb presses the semilunar bone backwards while traction and counter-traction are applied. The hand and carpus, which is dorsiflexed on the radius and ulna, is suddenly plantar flexed.

Treatment: If the case is examined early, reduction under a general anaesthetic can usually be accomplished. See Fig. 1. Reduction after some weeks may be impossible and open operation through an anterior incision is necessary. Sometimes the bone is found ischaemic and complete removal becomes necessary. The results can be excellent.

THE WRIST

Sprains of the carpal and wrist joints

These joints may be considered together, as in a sprain of the wrist joint the intercarpal joints are bound to suffer injury as well.

Mennell[2] has shown that, in dorsi-flexion (extension) and palmar flexion of the wrist some of the movement takes place at the intercarpal level, between the lunate and the capitate.

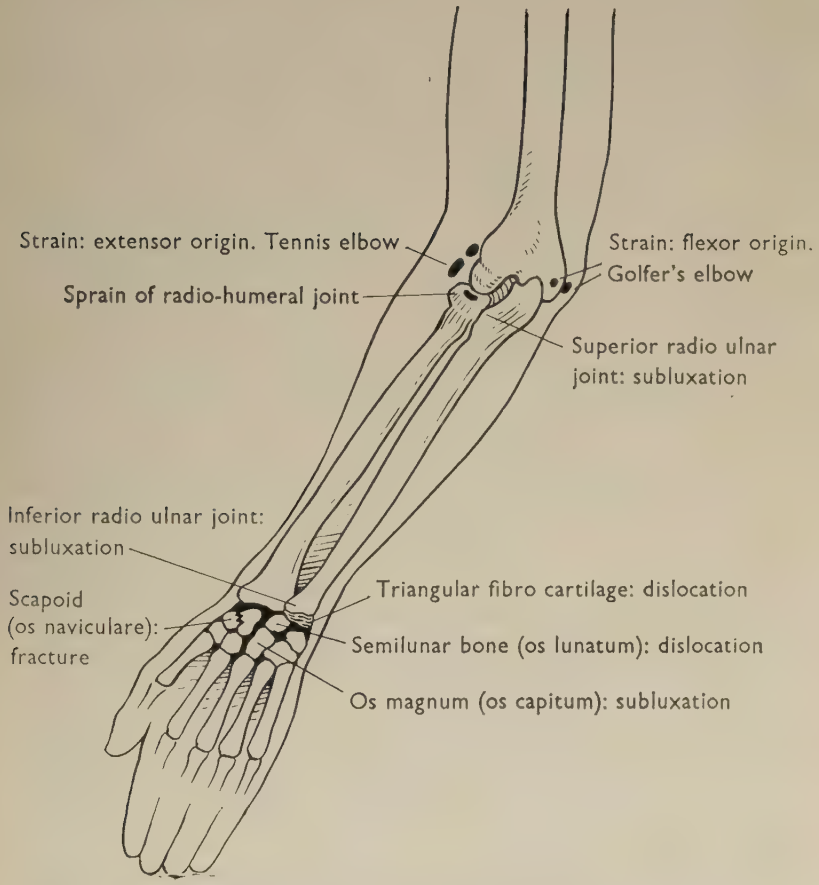
Strain: extensor origin. Tennis elbow
Strain: flexor origin. Golfer's elbow
Sprain of radio-humeral joint
Superior radio ulnar joint: subluxation
Inferior radio ulnar joint: subluxation
Scapoid (os naviculare): fracture
Triangular fibro cartilage: dislocation
Semilunar bone (os lunatum): dislocation
Os magnum (os capitum): subluxation

FIG. 2. Various anatomical entities involved in injury of the elbow and wrist.

It is imperative to take radiographs in four planes—anteroposterior, posteroanterior, lateral and oblique—and if possible, comparative ones of the opposite side. The reason for this is to exclude a fracture of the carpal scaphoid or dislocation of the semilunar. It is possible to miss the fine line of fracture of the scaphoid in the early stages, and in all cases of sprained wrist, a

check radiograph should be taken in three weeks if the first ones show no fracture.

The injury is nearly always caused by a fall. If it is the thumb side (the thenar eminence) which takes the force of the fall, the radial side of the wrist, which includes the scaphoid is affected; whereas, a fall on the little finger side (hypothenar eminence) is liable to affect the triangular fibro-cartilage between the distal ends of the radius and the ulna forming the inferior radio-ulnar joint above, and the triquetrum and pisiform bones below. An injury caused by a blow at boxing or a hand off at rugby will have the same effect.

Acute sprains require urgent treatment along the following lines: protection and support; felt and a crepe bandage or padded removable plaster cast; rest from strain but graduated exercises; home treatment; and physiotherapy.

If the patient is not progressing satisfactorily, an injection of hydrocortisone may be indicated.

Physiotherapy should include friction massage as deep as the patient can stand. Some cases of sprain of the intercarpal joints are slow in healing because there has been involvement of the opposing articular cartilages, which become rolled at the edges. They respond in time to conservative treatment without operation.

An example of this slow healing with eventual recovery was observed in a batsman, who sustained a direct blow on the base of the left thumb from a cricket ball. Radiograms taken on three separate occasions showed no bone injury. When seen a month after injury, he had slight swelling of the intercarpal joints between the proximal scaphoid and lunate and the distal trapezium and trapezoid. The batsman stated that each time he hit a ball he experienced excruciating pain at the base of the thumb and this caused him to drop the bat. The diagnosis was contusion sprain, with involvement of the articular cartilage of these carpal joints. It was important to get him symptom free as soon as possible, but in spite of physiotherapy for four hours a day, he needed twenty-five days' intensive treatment combined with home treatment before he could resume play. During the first period, the affected joint tissues swelled to the size of a golf ball and became exquisitely tender. The peak of the swelling was about the twelfth day. After the twelfth day it gradually resolved and the symptoms disappeared, but during the stage of aggravation the patient had to take strong analgesics to sleep.

Contusion sprains involving the triangular fibro-cartilage are equally difficult to make symptom free, and often require a long period of intensive treatment. Sprains of the wrist joint, especially in young girls, sometimes appear to be associated with rheumatic tendency, and to be part of the arm-shoulder girdle syndrome (thoracic outlet syndrome). They often require prolonged

physiotherapy and carefully graduated exercises. Some sort of wrist support is essential, especially when the patient resumes games. It is necessary to treat the whole arm, with remedial exercises to all the muscles up to the shoulder girdle and cervical levels.

Tenovaginitis

On the dorsal aspect of the wrist joint there are five separate compartments and tendon sheaths:—

1. Abductor pollicis longus and extensor pollicis brevis
2. Extensores carpi radialis longus and brevis
3. Extensor pollicis longus
4. Extensor digitorum communis and extensor indicis
5. Extensor digiti minimi
6. Extensor carpi ulnaris

All these tendons with their covering sheaths are liable to inflammation. Nissen[3] describes how the amount of synovial membrane varies in individuals. Those patients developing tenovaginitis seem to possess larger amounts than the average individual. It would seem that an allergic state, some toxic focus or chronic repeated strain are factors in the production of tenovaginitis.

De Quervain's tenovaginitis of the extensor tendons of the thumb and wrist: The tendons usually involved are the brachio-radialis at its insertion, the abductor pollicis longus and the extensor pollicis brevis. The extensor carpi radialis longior and brevior are sometimes involved near their point of insertion. It is, however, the inflammation of the tendon sheath of the second and third tendons which gives rise to difficulty in treatment. Some cases clear up with the elimination of a septic focus, rest from strain and physiotherapy consisting of friction massage and interferential therapy. It would seem that hydrocortisone injections are often helpful. It must be remembered that these cases pass through acute, sub-acute and chronic stages, and the result of conservative treatment, therefore, varies. Frequently, however, the case presents as a chronic state in which several forms of physiotherapy have been tried without avail, and operative freeing of the tendons from the constriction in the sheath has to be undertaken. At least half of the constricting part of the sheath must be removed, and the surgeon should take care that no restriction is left either at the top or bottom ends. Operative results are usually excellent.

Tenovaginitis of the extensor carpi-radialis longior and brevior occurs some $1\frac{1}{2}$ in. above the styloid process of the radius, either alone or in association with de Quervain's tenovaginitis. It is usually amenable to conservative treat-

ment such as hydrocortisone injections followed by physiotheraphy and home treatment. The wrist is supported from strain but the patient carries out graduated remedial exercises.

The long abductor and short extensor of the thumb may develop inflammation as a result of strain from constant practice at golf, tennis or cricket. It occurs over the muscular bellies of these muscles some $3\frac{1}{2}$ in. above the wrist joint. In the early stages silky crepitus can be felt. If the only treatment given is rest and support, for example, in a plaster cast, as soon as the support is removed the condition may recur. But, if a removable plaster cast is used to support the wrist, and hydrocortisone injections, home treatment and physiotherapy are given, the condition often resolves within a few days. If it is allowed to become chronic, so that the sheath of the muscle becomes thickened, recovery may be delayed several weeks.

The extensor or flexor carpi ulnaris is often involved in an inflammatory, sclerosing type of tenovaginitis especially affecting the left wrist, particularly in a golfer. The tenovaginitis may be acute as the result of the checking of the left wrist when the club head hits the ground. These muscles together are the main ulnar deviators of the wrist. The subacute or chronic stage occurs more usually as the result of continual false production of the shot, or of continually taking a divot when the ground is hard. Treatment is similar to that for the other forms of tenovaginitis—conservative at first and if this fails operative.

THE FOREARM AND ELBOW

Contusion of the tissue of the forearm

The efficacy of active treatment is illustrated by the following case. One afternoon a cricketer was hit on the back of the forearm, where a very considerably haematoma developed. Physiotherapy had been given to reduce the swelling which, when seen that same evening, was still considerable and causing intense pain through tension. The haematoma was situated in the middle of the back of the forearm where a small incision was made under local procaine anaesthesia and a large amount of blood evacuated. A further injection of 5 ml. of 2 per cent procaine was injected with hyaluronidase and a firm pressure bandage applied. The patient was instructed to remove the bandage before going to bed in order to give the arm contrast baths, and to repeat the baths the next morning. On examination the following day, sixteen hours after the injection, there was hardly any swelling at all, and indeed recovery had taken place to such an extent that the cricketer was able to play that day, and played in a Test match a week later.

Tennis elbow

This term covers several separate entities. It occurs primarily as the result of progressive strain of the forearm muscles of which the extensors and supinators of the forearm are the first to be involved.

In the action of extending the elbow and simultaneously dorsi-flexing the wrist and supinating the forearm from the pronated position, the muscles are acting against gravity. If to the weight of the hand is added a racquet or some other instrument, the muscles producing these actions may become strained. The strain will show itself as tenderness at three definite points over the lateral humeral condyle: (i) the origin of the extensors from the lateral humeral epicondyle: (ii) over the radio-humeral ligament: (iii) over the head of the radius and orbicular ligament. In time the superior radio-ulnar and radio-humeral joints become involved. This accounts for the typical symptoms localized to the outer side of the elbow joint. The symptoms may appear suddenly following a certain definite movement, or come on gradually getting worse if the patient persists with the exercise involving the action of elbow extension with dorsi-flexion of the wrist and supination of the forearm from the pronated position, i.e. turning the palm of the hand from the down position to the up, open position. In time, the antagonist group, i.e. the flexor group of muscles and the pronator radii teres over the opposite medial condyle become tender, probably because of unbalanced action between the extensors and supinators and the flexors and pronators.

Now the main supinator of the forearm and flexor of the elbow joint is the biceps brachii which is brought into action and extensively employed in the movements associated with tennis elbow. The action of turning the hand over from the palm downwards position to the palm upwards position is usually started with a certain degree of flexion of the elbow and as the movement progresses the elbow becomes extended and supinated. Thus in almost all cases there is tenderness of the long head of the biceps brachii where it lies in the bicipital groove in the anterior aspect of the shoulder joint and the extremes of shoulder movements are limited.

This complex action of the forearm is employed in the back hand tennis shot; and hence the name 'tennis elbow'. At the end of the nineteenth century the forehand shot was made with the head of the racquet dropped, so that the forearm became pronated starting from the supinated position. The 'tennis elbow' produced by this action showed itself first of all as tenderness of the structures at the origin of the flexors and pronators radii teres from the medial humeral epicondyle and medial collateral ligament.

Symptoms of strain of the extensors and supinators occur from many actions apart from tennis and are common in many other types of sport.

Strains of the flexors and pronator radii teres on the inner side of the elbow can also occur from any action in which there is lifting or pulling of objects from the supinated position. It occurs in the right elbow of right-handed golfers. As the right-handed golfer hits against the resistance of the extended and fully supinated left forearm, he may also get symptoms of tennis elbow on the outer side of the left elbow.

If the symptoms involving either the extensors or flexor muscles persist, in time the elbow and superior radio-ulnar joints become involved, first of all with inflammatory thickening of the synovia, and articular cartilage erosions which may eventually lead to osteoarthritis.

Strain of the forearm muscles may be part of a general strain of the whole of the arm, and can be part of the syndrome associated with the thoracic outlet and cervical spondylosis. The symptoms may start after the forearm muscles have been misused. They begin with pain and tenderness appearing at the origin of the extensors and supinators of the forearm with later involvement of the flexors and pronators. Gradually the condition of strain extends to the muscles of the shoulder, shoulder girdle and cervical vertebral joints. In other cases, the symptoms may start in the neck and shoulder with a break-down from strain of the muscles of the cervical vertebral joints and shoulder girdle, gradually working downwards to the hand.

Furlong's (1953)[4] contribution has previously been mentioned. It would seem that progressive strain due to postural and occupational causes produces vascular degenerative changes and hence fibrosis in the origin of the muscles.

Symptoms: the main symptom of tennis elbow is pain, which may be so severe, when the forearm is used for any movements more complicated than simple flexion and extension, that the patient may drop even the lightest article. The pain may persist as an ache, and, in the acute phase, may be sufficient to keep the patient awake at night.

Points of tenderness: the main points of tenderness are over the lateral epicondyle, the radio-humeral, and orbicular ligaments on the outer side, the medial epicondyle and the medial collateral ligament on the inner side, the olecranon fossae at the back of the elbow, and the long head of the biceps brachii in front of the shoulder joint. Movements of the elbow are usually limited: full extension may be limited by one or all of the following:—shortening of the extensors and flexor muscles of the forearm from both sides of the humerus; spasm of the biceps brachii; granulation tissue in the olecranon fossa.

If tennis elbow is associated with the thoracic outlet syndrome, such symptoms as pins and needles, numbness and tingling in the hand, a feeling of

bursting due to swelling of the hand and forearm, may be experienced and even severe cramps and pains in the muscles of the hand and forearm. Pain may radiate down from the neck or be localized to the course of the dorsai interosseus nerve.

It is possible to distinguish six different types of tennis elbow.

Type 1. The acute strain type in a patient under thirty with a definite history of injury. The extensor carpi radialis brevior is the muscle principally involved.

Treatment by hydrocortisone injection, home treatment and physiotherapy with the appropriate period of rest from strain usually produces a cure in two to three weeks. The hydrocortisone injection may have to be repeated on several occasions.

If the patient is first seen some months after the onset of symptoms, the condition often responds to one manipulation. This has the effect of breaking down adhesions formed in the origin of the muscles as the result of a muscle tear, and accounts for the miracle cures often claimed by bone setters and osteopaths.

Type 2. The type which comes on gradually and insidiously, without a definite history of injury, usually in patients over thirty-five. This type may

FIG. 3. Manipulation for tennis elbow. The elbow joint is extended fully and the wrist is pronated and palmar flexed forcibly, so as to stretch the wrist extensors. At the same time the pressure is so exerted to cause the forearm to take up a position of cubitus varus.

be occasionally complicated by the presence of a marked periostitis of the lateral epicondyle or a plaque of calcification in the soft tissue just lateral to the epicondyle. (Osgood's Bursa.)

The results of treatment depend on the stage at which the condition is first treated. A quick recovery will occur if the positive phase or chronic stage of healing has been reached; a slow one is likely if the case is seen when in the negative phase or acute stage.

Hydrocortisone injections given at weekly intervals over the three points on the lateral side of the elbow until the patient is symptom free. Manipulative movements of the elbow, shoulder girdle, and cervical vertebral joints are an important part of daily physiotherapy.

Treatment must include deep friction massage and even xylocaine and hydrocortisone injections to the long head of biceps brachii at the shoulder joint level, but restoration of the extensibility of the muscle throughout its whole course and length must be obtained by appropriate physiotherapy including gentle manipulations.

FIG. 4. Manipulation of the elbow. The head of the radius is held between the manipulator's thumbs. The radio-humeral joint is gapped laterally and hyperextended. The patient's hand being held in the operator's axilla.

In resistant cases, a manipulation under full anaesthetic may be necessary. The manipulative technique is important. The elbow is flexed with the forearm in the fully pronated, then the neutral and finally the fully supinated positions. It is manipulated into extension as in the Bankart Manoeuvre, i.e. the surgeon extends the elbow fully and at the same time forcibly pronates the forearm and wrist and flexes the wrists and fingers. Another way of breaking the adhesions on the outer side of the joint is to extend the elbow fully and at the same time open the outer side of the joint by placing on it a varus strain at the elbow. The olecranon may be moved from side to side in its fossa by rocking the forearm from side to side with the elbow supinated and slightly flexed. An adhesion can often be felt to give in the elbow when the arm is internally rotated and placed up the back. After the manipulation of the elbow joint, it is important to ensure full movements of the wrist, shoulder, shoulder girdle and cervical spinal joints. Finally, after these have been put through their full range, the elbow joint is again checked to see that the movements are full. On starting exercise strapping support to the wrist and elbow is helpful.

Occasionally one may have to remove a plaque of calcified tissue (Osgood's Bursa) or periosteal thickening.

In all cases the inner side of the elbow joint is included in the treatment. During treatment the area of tenderness on the outer side may spread down the muscles, even as far as the muscles over the dorsal interosseous nerve. It may even be helpful to inject hydrocortisone and local anaesthetic into these tender muscles to relieve the symptoms. Garden[5] has described operative lengthening of the extensor carpi radialis brevis in the lower forearm for resistant cases of this type. His views on the subject are interesting and include the following:—

1. Tennis elbow is a condition usually of middle age and degenerative changes in the orbicular ligament with contraction of the extensor carpi radialis brevis, may underlie its pathology.

2. Those cases not relieved by conventional treatment are often benefitted by Z-lengthening of the extensor carpi radialis brevis which neither causes any diminution of the power of wrist dorsiflexion nor efficiency of grip.

Types 3 *and* 4. Similar conditions to 1 and 2 appear on the inner aspect of the joint, affecting the origin of the flexors and pronator radii teres. The treatment of both conditions is along the same lines as in 1 and 2. The ulnar nerve may become involved and symptoms of ulnar nerve pressure appear. These types form the present day golfer's elbow or the old form of tennis elbow.

Type 5. Tennis elbow associated with osteoarthritis of the elbow joint, with involvement of the superior radio-ulnar and radio-humeral compartments of the joint. This often develops in the older athlete as an extension of Type 2.

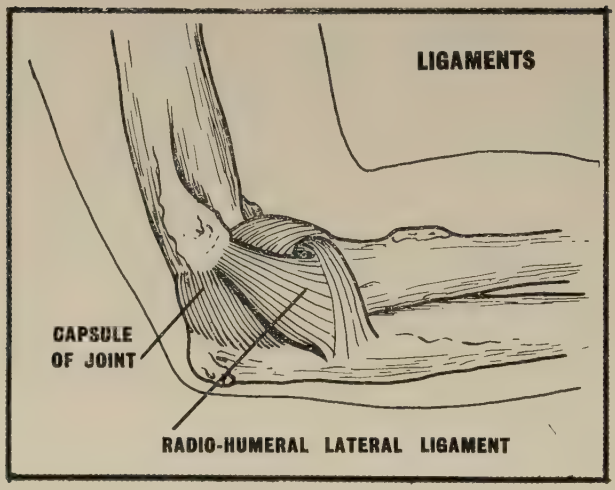

FIG. 5. Tennis elbow involves a strain of the extensor muscles that sweep down from the external epicondyle at the elbow. Also the radio-humeral ligament is strained. There is tenderness of the arm and any lifting or gripping causes pain, especially with the forearm turned palm down.

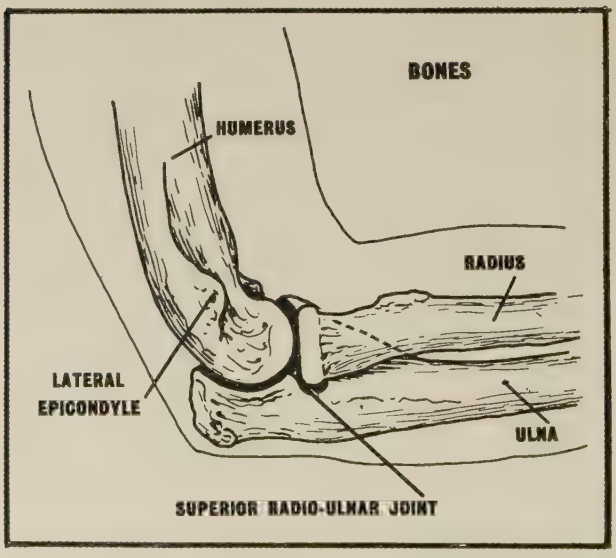

FIG. 6. In changing position from palms down to palms up there is usually some jarring against resistance so that the supinator brevis muscle, the superior radio ulnar joint, and also the radio-humeral compartment of the elbow are involved.

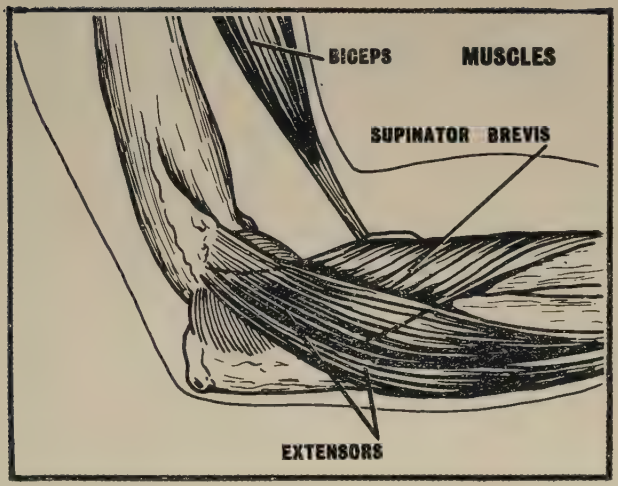

FIG. 7. In cases that have gone on for some time the extremes of extension and flexion are limited and painful if forced. Often this limitation of movement is accompanied by spasm of the biceps. This muscle is important in steadying the elbow.

Treatment follows the lines already described. It may have to be both regular and prolonged. If the condition does not respond, deep X-ray therapy or even arthrotomy may be necessary. Trethowan[6] described synovectomy of the superior radio-ulnar and radio-humeral compartments of the elbow joint for certain cases of this type. Another approach which relieves the symptoms in selected cases is operative freeing of the extensor origin from the lateral epidondyle. The thick fascia over the extensor muscles is incised in order to release the radial nerve and its dorsal interosseous branch. (A.14.)

Often cases of this type remain relatively symptom free if they carry out regular home treatment and modified activity. The shoulder girdle and cervical vertebral muscles should all be included in the remedial exercises.

The views on tennis elbow of Dr. Henrique de Goés[7] of Brazil are interesting. It would seem that his operation is suitable for Type 2 and those cases which have extended to Type 5. He thinks tennis elbow is a pathological condition stemming from an anatomical predisposition. Traumatic conditions produce hypertrophied capsular folds and synovial fringes. Sometimes this thickening is meniscus-like and its removal produces a cure.

Type 6. Cases associated with the thoracic outlet syndrome, cervical spondylosis, acroparathesia or median nerve compression in the carpal tunnel. These patients require rest from strain, returning to full use of muscles care-

fully and gradually. Operative removal of a cervical rib or the release of the contracted scalenus anticus and medius muscles sometimes improves the symptoms. Pridie[8] has noticed that many of his patients with cervical disc degeneration have symptoms of this form of tennis elbow, and that the condition is cured by anterior fusion of the vertebrae associated with the degenerative disc.

On the whole, however, this type of case usually improves with periodic courses of treatment, especially if home treatment and postural training are carried out indefinitely.

Our views on strains of the elbow can be summarized as follows:—

1. Anatomical abnormalities which predispose to pain.

(a) We would agree with de Goés that certain people have anatomical abnormalities of the elbow joint, such as a contracted extensor carpi radialis brevior, or a tight orbicular ligament.

(b) Abnormalities in relation to the thoracic outlet, such as a cervical rib or abnormalities in the axillary inlet.

2. The wrong use of the forearm muscles. This means that the activating forearm muscles are not being worked on firmly adjusted synergic and prime fixing muscles. If the thousand and one actions of the hand and forearm muscles which the individual has to carry out during the day, are performed with the shoulder girdle and shoulder slumped, mechanical strain is bound to occur.

3. Symptoms are only experienced when grasping articles with the wrist fixed. As well as the elbow they sometimes occur in the wrist and shoulder. Attention to the size of the racquet handle and suitable support for the wrist and elbow are important adjuncts for tennis players.

4. The symptoms may be produced by prolonged faulty overuse. As to whether the primary symptoms are on the inner or outer side of the elbow joint depends on which group of muscles is being primarily strained.

5. Septic foci (from teeth or tonsils), or a gouty diathesis can be predisposing causes.

6. Operative methods may be necessary in a few cases but a thorough period of conservative treatment should be given first. This should include rest from strain, exercises without strain, hydrocortisone injections, various forms of physiotherapy including manipulation with or without anaesthetic, and treatment of the whole arm as an entity up to the shoulder girdle and neck levels.

7. The symptoms of the various forms of tennis elbow are often associated with cervical spondylosis.

'Pulled' and 'Pushed' elbow

Mennell[9] describes these conditions as separate entities. As the result of a wrenching strain on the hand, the head of the radius is forced down on to the orbicular ligament and the locking effect of this ligament and the ligaments of the inferior radio-ulnar joint prevents the normal free play of the radius up and down on the ulna, and causes symptoms of 'pulled' elbow.

'Pushed' elbow results from injury such as a fall on the outstretched hand: the head of the radius is forced upwards and the opposing articular surfaces may have a secondary effect on the inferior radio-ulnar joint. Symptoms of pain, loss of power, and limitation of movement, may be present in both the elbow and wrist joints.

The early acute phase of these conditions should be treated by manipulation, force being applied in the reverse direction to the force of injury. In 'pulled' elbow the surgeon pushes the radius upwards: in 'pushed' elbow he pulls it down. Physiotherapy and home treatment are required. As the radius in full pronation glides forwards on the ulna it is important in manipulation to establish full anterio-posterior movement of the radius on the ulna before forcing rotation.

If the conditions are allowed to become chronic the interosseous membrane between the radius and ulna becomes contracted and thickened, and so do the structures of the elbow and inferior radio-ulnar joints. Recovery is slow. Treatment should consist of a series of injections combined with intensive physiotherapy and home treatment. Manipulation and massage are an important part in physiotherapy in order to stretch the interosseous membrane. Sometimes a manipulation under anaesthesia is required.

Dislocation of the elbow joint

This may be simple dislocation, or dislocation associated with fractures and other complications.

In simple dislocation, if the X-ray is studied minutely one sometimes can recognize small flakes of bone near the epicondyles, particularly the external. The dislocation should be reduced with the greatest care, preferably under a general anaesthetic and in a sterile operating room, so that, after the reduction, small incisions can be made over the epicondyles in order to express the haematoma. Sometimes small fragments of bone with muscle attached can be expressed through the wound. After reduction, the surgeon applies gentle pressure to make sure that the dislocation is stable. A well padded back slab is put on with the elbow at a right angle, and the elbow is maintained in the padded plaster for two days. Movements of the fingers and gentle movements of the elbow, including flexion and extension against the resistance of

the plaster, are started at once. At no time must these movements be other than active, without strain and without force. After 48 hours the posterior slab of plaster is removed, and the elbow is soaked in a weak solution of warm Dettol daily. This allows seepage of further products of traumatic effusion through the small incision on the inner and outer side of the elbow.

Gentle flexion and extension exercises of the elbow, with pronation and supination exercises are allowed, and should be done twenty times slowly during the interval of the soak, which should last twenty minutes. The part is dried with a sterile towel and protected with gauze and a crêpe bandage. The elbow is returned to the posterior slab, which is held in position by a further crêpe bandage. At this stage very cautious and gentle faradism may be given to the arm and forearm muscles.

This régime is carried out daily, but the patient is allowed to remove the plaster for a further period each day to perform gentle remedial exercises without soaking. By the end of the eighth day the small incisions should be soundly healed. From now on physical treatment is continued. Wax baths, anodal galvanism or shortwave, with gentle faradism, combined with home treatment, are given. The posterior slab is maintained for four weeks, being removed twice daily for the treatment. It will be found that by the end of a month the movements of the elbow are normal, and, gradually, over the next six weeks, more and more strain is permitted.

Home treatment should continue to be carried out conscientiously twice daily, and organized physiotherapy three times a week at first and then twice a week.

It may be possible to allow the patient to use the elbow for light duties at the end of six weeks, but not before. Most simple dislocations will have made almost a full recovery by this time.

The object of evacuating blood clots and the tiny pieces of bone is to ward off myositis ossificans. With this object in view, also, the utmost gentleness is used throughout the treatment. Nevertheless, despite these precautions, traces of myositis ossificans may appear: but the active gentle treatment reduces the severity of the condition and helps to restore elbow function as soon as possible.

Dislocation associated with fractures may be stable after reduction, or unstable. The stable ones are treated as already described except that the initial padded plaster back slab is maintained for a week. A complete, encircling plaster should never be applied to any elbow injury likely to be followed by appreciable reactive swelling. During this time the patient should practice active movements against the resistance of the plaster and receive gentle fara-

dic contractions to the arm and forearm muscles. At the end of a week, the elbow is soaked daily for seven days in a weak antiseptic solution, and then treatment continues as for a simple dislocation.

The unstable dislocations may be so complicated by a comminuted fracture that no form of metal fixation is possible. These are only maintained in a relatively good position by firm plaster fixation, which must be maintained until the fracture is well enough consolidated to hold the fragments in place. This takes about six to eight weeks, and during this time active isometric movements against the resistance of the plaster are encouraged, and faradism to the arm and forearm muscles given. Movement in this type of case is usually regained slowly but is considerably helped by conscientious home treatment and regular physiotherapy. It is worth continuing with twice daily home treatment and with physiotherapy twice a week for twelve to twenty-four months.

In grossly comminuted fractures of the lower end of the humerus reduction and stability are sometimes accomplished, especially in young children, by gentle skin traction applied with the elbow in extension. At the end of two weeks a moulded plaster is applied, if necessary under a full anaesthetic. The traction is maintained for a further week, and then, for a fornight, the patient carries out strong isometric exercises with the elbow in the plaster. After five weeks the fracture-dislocation should be completely stable and exercises, especially flexion ones, may commence with the elbow out of plaster. Gradual improvement takes place over the next two years. At no time should movements be forced.

If a large fragment of bone is separated, fixation by open operation employing a screw is necessary. Plaster is maintained for three weeks and then the case treated on the same lines as for a simple dislocation.

Complications of dislocations, such as involvement of the blood vessels and nerves, particularly of the ulnar nerve, are watched for carefully and proper treatment instituted immediately they appear.

Those interested in baseball injuries of the elbow joint should refer to Chapter 4, p. 148 where a resumé is given of the late Dr George Bennett's work on this subject.

The Javelin Thrower's arm or elbow

Miller[10] points out that the condition of the javelin thrower's elbow is more common than was generally supposed and describes two types: one due to throwing round arm, which causes a strain of the medial collateral ligament: and one through throwing overarm, the correct method of throwing the javelin. This leads to roughening and new bone formation of the tip of the

olecranon or, in some cases, to actual avulsion fracture of the tip of the olecranon.

The first type can be relieved temporarily by the injection of a local anaesthetic, and hydrocortisone injections may produce temporary or complete relief. In the second type rest is most important but the condition tends to recur. If there has been an avulsion fracture of the olecranon the condition is completely relieved by the removal of the bony flake and reconstruction of the triceps attachment.

THE UPPER ARM

Contusions and sprains of the muscles of the arm are treated actively as advised in Chapter 10.

Rupture of the long head of biceps occurs if the elbow is flexed when the forearm is suddenly checked by any means, such as an opponents arm or when there is a heavy weight in the same hand. Operative repair may be considered. The ruptured tendon is sutured, or the distal portion sewn down in its groove and the upper portion removed.

The following are three illustrative cases of the condition.

A young rugby footballer aged twenty was jumping for the ball in the line-out when his arm was suddenly checked by the arm of another player which caught his forearm. When he was seen two days later there was pain, swelling and tenderness over the biceps bulge in the upper arm. It was obvious that the patient had sustained a rupture of the long head of the biceps brachii. The tendon was sutured below the bicipital groove under the fascial expansion of the insertion of pectoralis major and latissimus dorsi.

An acrobatic dancer, aged forty, had experienced considerable pain some three months previously, while he was holding his partner in a long arm balance. At the time of the accident a diagnosis of brachial neuritis had been made as there was much pain. When he was first seen by me, it was obvious that, although he had sustained a rupture of the long head of biceps brachii, his symptoms were typical of tennis elbow because of the additional strain on the extensor muscles of the forearm. With active treatment, including home treatment and physiotherapy the patient was able to continue with his acrobatic act in three weeks.

On the strength of this success, a second acrobatic dancer, also forty years of age who was also seen on the day of injury, was treated by active conservative treatment and was able to perform a full television programme exactly three weeks from the date of injury.

In neither of these cases had the neighbouring muscles been allowed to be-

come wasted and atonic: and in fact by graduated exercises they had hypertrophied and thus were able to take on the duty of the ruptured biceps.

INJURIES TO THE SHOULDER JOINT

The shoulder joint, being exposed, is particularly prone to direct injuries from blows or falls or to indirect injuries from a fall on the outstretched hand or from a quick movement of the arm. In the shoulder joint the tendinous fibrous cuff of the external rotator muscles is inserted in close relation to the attachment of the capsule, in the vicinity of the greater tuberosity laterally and superiorly. These muscles fix the head of the humerus and allow the overlying deltoid muscle to abduct the arm. If they are injured, the head of the humerus cannot be securely fixed, so that the movement of abduction is

FIG. 8. Manipulation of the shoulder joint. To demonstrate the importance of pushing the head of the humerus well down into the glenoid cavity when manipulating the shoulder joint. The uppermost hand is pushing the head down while counter pressure is exerted by the hand on the under side of the arm pushing up. Traction is applied while this is being carried out. The hand of the assistant in the axillary space exerting countertraction cannot be seen.

FIG. 9. Manipulation of shoulder. Abducting the arm. The assistant's hand can be seen applying counter-traction. The head of the humerus is controlled by the right hand while the left hand slowly abducts the arm using a short leverage. The uppermost fingers of the manipulator's right hand can help to push the head of the humerus downwards and backwards into the glenoid cavity. The assistant's hand fixes the scapula.

limited or completely prevented according to the severity of the injury. Moreover if the arm is abducted passively above a right angle, it cannot be voluntarily kept there.

The long head of the biceps brachii is an intra-articular structure. It crosses the joint obliquely from its origin at the top of the glenoid fossa to the front of the greater tuberosity, and lies in the groove between this and the lesser tuberosity, where it is covered by the transverse humeral ligament.

Overlying the rotator cuff insertion and interposed between it and the undersurface of the acromial process and the deltoid muscle is the subacromial bursa. This bursa has two parts, the subacromial and the subdeltoid.

It can be seen that the structures most liable to injury are those exposed on the lateral, superior and anterior surfaces; that is to say the rotator cuff tendons, especially the supraspinatus; the long head of the biceps brachii; the subacromial bursa and the deltoid itself. Occasionally the structures arising from the coracoid process, the pectoralis minor, the coracobrachialis and the short head of the biceps are injured at the same time as the subscapularis and the pectoralis major.

In certain injuries, particularly dislocations, the circumflex nerve is most vulnerable, and may even become avulsed from the substance of the deltoid which is then permanently paralysed. Seddon[11] has shown that operative treatment has no effect because it is impossible to sew the nerve back into the muscle.

The shoulder joint is liable to an insidious type of capsulitis, in which trauma plays no part. Simmonds ascribes the condition to a degenerative lesion of the vessels of the paratenons. The tendons take their blood supply from vessels in the paratenons, and these are subject to degenerative changes such as endarteritis in a young patient and arteriosclerosis in the older one. Both conditions subsequently lead to fibrosis of the tissues involved. It usually occurs in patients over forty and is associated with cervical spondylosis particularly where there are degenerative disc lesions of C5, C6 and C7. American authors have described the neck, shoulder joint and hand syndrome in which a capsulitis of the shoulder joint is accompanied by a swollen painful hand and symptoms of acroparaesthesia. Dupuytren's contracture, stenosing tenovaginitis and compression of the median nerve in the carpal tunnel may occur in association with this syndrome.

Capsulitis may arise, particularly in the older athlete as the result of a trivial injury such as changing gear in a car or putting out an arm to make a stroke at tennis or golf. It seems probable in these cases that there is a de-

generative lesion already present and movements of the joint have already become restricted by adhesions. The patient, being conscious of pain, considers he has strained the joint. Possibly each time he makes the action or a similar one, an aggravation occurs with the outflowing of a plastic exudate. This organizes into further adhesions, so that there is a gradual diminution in the range of movement. The restriction of shoulder movement has often to be over 50 per cent before the patient is conscious of it. He can often overcome the diminution in movement by twisting and sidebending his body subconsciously, and it is not before abduction is restricted to less than a right angle that he becomes aware of the disability.

FIG. 10. Manipulation of shoulder joint. Traction on the abducted arm is applied by an assistant so as to pull the head of the humerus away from the glenoid cavity. Countertraction is not shown in this case. The manipulator now has both hands free to palpate the bones. In this case the right hand controls the humeral head while the left hand pushes it downwards, backwards, forwards, and upwards, finally rotating it.

Saha (1958)[12] has pointed out there are three main types of movement in the shoulder joint:—abduction and adduction of the arm around a fixed pivot in the middle of the glenoid; rotating movements lateral and medial around the same fixed point in the glenoid: and a gliding movement of the head of the humerus so that the head can be moved forwards, backwards, upwards or downwards in the glenoid cavity. These gliding movements are essential for full movement and full function.

We have already noted that, in 'tennis elbow' the long head of the biceps

brachii may be involved. There is often a concomitant mild capsulitis of the shoulder joint.

The following are the main types of injury to the shoulder joint:—

1. sprain
2. supraspinatus strain, tear or rupture
3. subacromial bursitis
4. lesion of the long head of biceps brachii
5. subluxation and dislocation
6. traumatic capsulitis
7. periarthritis (capsulitis leading to frozen shoulder).

Following an injury and sometimes an apparently trivial one, the shoulder is often maintained continuously in a sling at the side and completely rested; with the effect that adhesions form and a traumatic capsulitis starts. Perkins stresses that, even in fractures, the soft tissues are as important as the bone and treatment of them should in many cases take precedence over the fracture, in order to avoid the restriction of extremes of movement which comes from traumatic capsulitis.

Ellis[13] recommends modified activity from the outset, because the proteins in the traumatic effusions bind down the long head of the biceps brachii in its groove and scapulo-humeral movements quickly become limited.

Diagnosis

Apart from an accurate history and examination, there are additional tests which may help. Antero-posterior radiographs should be taken with the arm internally and externally rotated, and lateral views are also desirable. All the pictures should be compared with corresponding ones of the opposite side. Special comparative films are taken from above. Radiographs after the injection of opaque substances are helpful in differentiating partial and complete tears of the supraspinatus tendon and involvement of the subacromial bursa, but should not be undertaken lightly as they often give rise to considerable pain. Differential electromyelograms are useful to give a prognosis in nerve injuries, particularly those involving the circumflex nerve.

The surgeon should note painful arcs of movement, both active and passive, with the palms up and down, and carefully compare these with the opposite side. The head of the humerus normally has free movement in the glenoid fossa. If the long head of biceps brachii is adherent in its groove, the anterior capsule becomes tight and prevents the head of the humerus gliding forwards and rotating inwards in the fossa: with the result that the patient cannot perform full horizontal extension or put his arm up the back fully. Plastic exuda-

tion forms adhesions and capsular fibrosis which bind down the head of the humerus to the external rotator cuff tendons, the overlying subacromial bursa and this latter structure to the deep surface of the deltoid itself, with the result that the head cannot slip downwards into the glenoid fossa and abduction is prevented or limited.

After injury the patient is often made to wear a triangular sling; and the result is that the long head of biceps brachii is firmly fixed in its groove in internal rotation, as this is the position of rest in the sling. External rotation becomes limited and so, therefore, does abduction, because until rotation is full, complete abduction is impossible. In posterior subluxations also external rotation is limited. Horizontal adduction can be tested by noting the position of the point of the elbow in relation to eyes, nose and ears. It is limited by plastic adhesions involving the postero-lateral part of the capsule, which prevent the humerus falling back in the glenoid fossa. It is important to establish exactly where the head of the humerus is obstructed by loss of capsular resiliency so that active treatment can be particularly directed to the area.

1. *Sprain*

No particular structure is specially involved, but there is swelling of the shoulder generally, with pain on movement.

There may be associated sprain, subluxation, or dislocation of the acromio-clavicular joint, and this equally requires treatment. Provided the shoulder joint is rested in a universal sling, taken out at regular intervals for active assisted exercises and other home treatment and physiotherapy, a mild sprain should recover in a few days, a moderate one in two weeks and a severe one in four weeks.

In some cases hydrocortisone or other types of injection may help to expedite recovery.

In the older patient recovery is naturally slower; but provided the right proportion of rest, active movement and treatment are given there should be no excuse for the development of 'frozen shoulder'—unless of course he is suffering from a mild degree of capsulitis and periarthritis associated with cervical spondylosis.

If every case is treated with the greatest gentleness, at the end of each day there should be a reduction of pain and increase of movement. If it appears that movements are becoming less and the muscles are beginning to stutter, fibrillate or fasciculate when an active movement is initiated, a focus of infection should be sought, especially in teeth, tonsils and sinuses. These should always be inspected at the first examination in every case of shoulder injury, no matter how young the patient.

2. *Supraspinatus strain, tear or rupture*

These injuries are all caused by the same type of mechanism: the degree depends upon the severity of the accident, which may be a sudden fall on the pronated hand, a check on the arm when it is being abducted or the force of a vigorous throw.

A strain should be symptom free in ten days with active treatment, but a slight tear may require three weeks constant daily treatment to recover, and a further three weeks gentle use before vigorous exercise such as rugby football is possible.

Tears of any magnitude and rupture should be operated on as soon as possible, especially in the active young athlete. Complete recovery is impossible without operation. After a fortnight of active treatment, operation should be considered at once in those patients who cannot maintain active abduction of the shoulder above a right angle.

The torn or ruptured tendon is repaired by silk or linen sutures. The removal of the acromion may have to be considered at the same time (vide infra). After the operation it may take as much as a year before the patient can return to a particularly vigorous sport. During this time he must undertake an active programme of rehabilitation.

Chronic supraspinatus strain often gives rise to the characteristic pain between 70° and 120° of abduction: after 120° the swelling of the tendon passes away from contact with the under surface of the acromium and pain ceases. Movement can now progress to the full unless the condition is associated, as it often is, with capsulitis; in which case there may be limitation of the terminal degree of movement.

In all types of supraspinatus injury the injection of hydrocortisone or other fluids in the early stages may help hasten recovery.

Calcified deposits are sometimes found in the tendon. They may be of considerable size, and may cause an acute excerbation with agonizing pain. Similar pain may occur during the actual formation of a deposit, for example during an attack of gout. It is worth having a serum or plasma uric acid test carried out so that specific antigout medication can be given.

If the pain is agonizing some cases respond to deep X-ray therapy in two to three applications but if this fails or is not available, operative removal of the chalky deposits gives instant relief. At operation the tendon at its insertion will be found swollen, red and under tension; and at the touch of the scalpel the chalky deposit spurts from the tendon and is easily removed. Acromionectomy is much the simplest and best surgical procedure for any of the four conditions which cause the so-called supraspinatus syndrome, but good results after it depend entirely on first-class rehabilitation.

Calcareous deposits are often found in the course of radiographic examination and may not themselves be the cause of pain because they may be demonstrated in the opposite shoulder which is symptom free. Sometimes they are associated with capsulitis of the shoulder and become symptom free with appropriate treatment of this condition. Occasionally pain on movement resists all forms of treatment. As well as a supraspinatus tendon tear there has been an associated fracture of the greater tuberosity or the tendon of insertion has been pulled away with a small fragment of bone. Cases of this type are cured by acromionectomy, but it is important to remove the whole of the acromion right up to the acromio-clavicular joint and in the same way follow the operation with a carefully graduated programme of exercises.

The following case illustrates the benefit obtained by the use of a combination of butazolidin and colchicine. The patient aged forty suffered from a painful, stiff left shoulder which prevented him carrying out his athletic activities. Radiographs showed massive deposits in the superaspinatus superiorly and posteriorly. Movements were restricted terminally and there was a typical painful arc starting at 70° abduction. Active treatment, including hydrocortisone injections on three occasions, followed by a manipulation under pentothal almost seemed to aggravate the condition and he was put down on the waiting list for admission. At this time it was found he had a high plasma uric acid; while awaiting admission he was put on a purin free diet and given a course of butazolidin 200 mgms. and colchicine 1/120 grain, one of each three times daily for a week. The following week he had butazolidin, 100 mg. with colchicine 1/120 grain t.d.s. for a week. The result was a completely free joint and operation was not necessary.

3. *Subacromial bursitis*

Due to its position between the insertion of the rotator cuff muscles and the under surface of the acromion process and deltoid muscle, this bursa usually suffers some degree of traumatic inflammation in sprains of the shoulder, supraspinatus lesions and dislocations.

Occasionally, especially in men with gout, an acute bursitis will appear following a trivial injury. It may give rise to excruciating pain and be the precursor of a periarthritis leading to a frozen shoulder. If the lesion is taken seriously, the patient can often be made symptom free in a few days. He must be rested in bed, given a universal sling and adequate analgesic and sedative medicines. Gentle physiotherapy, anodal galvanism, effleurage and minimal stimulation of the muscles by faradism, usually induce symptoms to settle. This scheme of treatment, which has been successful in hundreds of

cases, usually stops the inflammatory process in the early stages, thus preventing chronic capsulitis and frozen shoulder.

Occasionally calcareous deposits form in the bursa as well as in the supraspinatus tendon, but characteristically these are seen on a radiographic film as a fine line of calcification. This is found at a lower level (at the base of the greater tuberosity) than those in the supraspinatus. Occasionally, especially in the older patient the bursa may become chronically thickened and contain fibrinous bodies of varying sizes and number which require operative removal and even acromionectomy.

4. *The long head of biceps brachii*

Ellis[14] points out that the position of the long head of the biceps in a groove in front of the shoulder joint makes it very liable to be involved in any injury of the shoulder joint. The intra-articular tendon changes direction in its course just as it passes under the transverse humeral ligament, and at this point is liable to suffer injury by rub or by attrition. Wherever there is limitation of full extension of the elbow, there is a probability that the biceps brachii has shortened, with tenderness of the long head at the front of the shoulder. In some individuals, the angle of the lesser tuberosity is such that the tendon can jump from its groove. Unless the traumatic effusion of any injury is absorbed quickly, the proteins separate out and gum down the tendon and its sheath to form a stenosing tenovaginitis.

Hitchcock and Bechtol[15] state that lesions of the long head of the biceps brachii are one of the most common causes of pain and disability in the shoulder joint and are a distinct entity to be distinguished from acute and chronic subdeltoid bursitis, and injuries to the supraspinatus tendon and fibromuscular cuff.

Predisposing factors are:—

1. The presence of a ridge called the 'supratubercular ridge of Mayer'.

2. Variations in the depth of the bicipital groove. In other primates the medial lip of the groove always forms a constant angle; but in the human there is considerable variation from 90° to 15°.

3. The formation of an exostosis on the medial lip. In 100 human humeri examined, 8 per cent of cases had a complete supratubercular ridge and 58 per cent a partial ridge. In 45 per cent of these, a spur or exostosis was present on the medial lip of the bicipital groove. In the 33 per cent without a ridge only 3 per cent had evidence of an exostosis.

4. The orientation of the scapula. Phylogenetically in man the scapula has moved backwards and to the side of the thoracic cage; as the arm is used habitually in abduction and internal rotation, the tendon is forced forwards

and inwards on to the transverse humeral ligament and medial lip of the sulcus, and friction results.

In abduction and adduction it is the head of the humerus and the intertubular sulcus which alter their position in relation to the tendon. The patient complains of pain and tenderness along the line of the tendon in the bicipital groove down to the insertion of the pectoralis major. The pain sometimes radiates down the front of the arm. A clicking or snapping may be present when the arm is internally rotated in the abducted position. This can often be prevented by pressure over the groove.

This condition of the biceps brachii tendon has been recognized in this country for many years. There is often mild peritendinitis in the so-called tennis elbow where there is tenderness over the lateral humeral epicondyle, radio-humeral and orbicular ligaments. It accounts for some of the limitation of extension of the elbow joint in some cases of tennis elbow.

Active vigorous physical treatment including gentle daily manipulation will help to prevent shoulder injuries from becoming chronic. Several injections of 2 per cent Novocaine and hydrocortisone at weekly intervals around the tendon and into the sulcus, combined with vigorous physical methods including manipulation under full general anaesthesia will often cure chronic cases. Cases resistant to the above measures or those in which the tendon dislocates require operation.

If patients do not recover in a short time with manipulation and physical treatment, Hitchock and Bechtol recommend operation. An S-shaped incision is made over the delto-pectoral interval, the subacromial bursa and tendinous cuff are examined and the biceps tendon explored by incision of the capsule and transverse humeral ligament. Peritendinitis, adhesions of the tendon to the groove, laxity and buckling of the portion of tendon above the transverse humeral ligament, a shallow groove, a frayed tendon and exostosis on the medial lip—any of these may be discovered.

Hitchcock and Bechtol[16] recommend that tendinous adhesions should be freed, the sulcus deepened, and excision of the tendon above the transverse humeral ligament. Any exostosis should be removed and the distal portion of the tendon sutured to the deepened floor of the groove. The joint is then put through full range of movement to break down intra- and extra-capsular adhesions. After ten days, exercises are started and most cases show a complete recovery in eight weeks.

5. *Subluxation*

This occurs in the same way as dislocation but often happens only momentarily, for example in patients who have wasting of the shoulder muscles

following injury. The head of the humerus slips over the glenoid rim on a rotation abduction movement and usually reduces itself during the reverse movement.

Occasionally the long head of the biceps brachii slips out of its groove owing to damage to the transverse humeral ligament, or to the lesser tuberosity. The patient has the sensation of subluxation of the joint.

Some patients recover with appropriate active treatment, but a few require operative intervention to deal with the structural lesion. It is wise to give conservative treatment at least a month's trial unless it is obvious that the lesion cannot be cured except by operation. Sometimes a piece of the glenoid rim becomes separated and this causes an internal derangement of the joint and requires operative removal.

Dislocation

The shoulder may be put out by indirect or direct violence. The indirect injury occurs during a fall on the outstretched hand or elbow, and it causes a dislocation which may not recur: the direct injury may itself cause the dislocation, but it may be combined with indirect injury and produce a type of lesion liable to recurrent dislocation. When a footballer, for example, falls on the outstretched hand and at the same time is hit on the upper part of the humerus, the head of the humerus is forced downwards and inwards and the Bankart lesion may be produced. This consists of a stripping up of the capsular attachment to the glenoid rim, anteriorly and inferiorly and sometimes even a fracture of the rim. A furrow may also be ploughed in the superior aspect of the head of the humerus by contact with the glenoid rim. This can be observed on X-ray films taken with the arm in internal rotation.

Frequently there is an associated fracture of the greater tuberosity. Before attempting reduction, the surgeon should look for evidence of paralysis of the deltoid muscle or of radial nerve damage. If the patient is seen immediately after the accident, before the muscles have gone into spasm, it may be possible by gentle traction and manipulation to reduce the dislocation on the spot: but it is always best to have radiographs taken both before and after the reduction.

Anterior dislocation is far the more common, but sometimes the head of the humerus slips behind the joint. Posterior dislocation often happens in epileptics. The shoulder is usually held in internal rotation and intense muscle spasm prevents any attempt at producing external rotation. The only way to secure the relaxation necessary for reduction is deep general anaesthesia. When the humerus is back in place, the joint should be tested to see whether it is stable.

Occasionally it may be possible to reduce a dislocation some hours after the accident without much fuss or pain, if the patient is under the influence of morphia or pethidine.

After the joint has been checked by X-ray and a further examination made to exclude nerve injury, there are two courses of treatment open, each of which has its supporters. The first course is absolute immobilization of the shoulder joint for three weeks, in the hope that any capsular stripping or fracture of the glenoid rim will heal. Following this the patient regains movement by voluntary remedial exercises.

The second course is based on the conviction that immediate immobilization after reduction has no influence on the likelihood of recurrence of the dislocation, and, therefore, is a waste of time. Gentle active movements are encouraged from the first.

If after manipulation the reduction is found to be unstable, the arm must be immobilized to the side for three weeks.

Steeplechase jockeys often dislocate their shoulders. The following method of treatment usually enables the patient to return to riding within three weeks even if he has an undisplaced fracture of the greater tuberosity. After satisfactory reduction the elbow and forearm are supported in a universal sling. Gamgee is placed in the axilla and over the shoulder and held in position by two 6 in. crêpe bandages. Isometric active contractions of the shoulder muscles are started immediately, together with supported aided active exercises to the elbow, shoulder girdle and neck muscle. Effleurage should be given to the shoulder, shoulder girdle, cervical and elbow joint muscles over the bandages, in order to reduce muscular spasm and spread traumatic oedema. All the muscles are exercised three times a day, for five minutes, and the periods are increased one minute a day. The shoulder exercises at first isometric but later isotonic are against the resistance of the bandage. As soon as possible measurements for a check strap are taken. The bandages are removed once each day for effleurage, gentle faradism and assisted active movements to the point of the beginning of conscious restriction. In this way no pain is produced and after each treatment there is a definite increase of movement. For three weeks the movements are restricted to those below a right angle and the check strap is employed in riding. After a few days the universal sling can usually be left off for increasing periods, so that the patient can perform rehabilitating exercises. Riding, hacking or exercising is often possible with the use of the check strap at the end of a week.

The sling is maintained for short periods over three to six weeks, so as to prevent the shoulder muscles becoming tired or strained, but many cases so treated have full movement and power by six weeks. As improvement takes

place physiotherapy is dropped, but active home treatment should continue for many months to ensure that the joint movements are restored to full.

Other athletes can be treated in the same way. Complications, such as fractures of the greater tuberosity, lesions of the supraspinatus and the long head of biceps brachii tendon or associated nerve involvement make recovery slower. If recovery appears to be unduly slow, therefore, the surgeon should look for one of these lesions, for an undiagnosed posterior subluxation, or for a septic focus. Even after recovery of full movement and power, the muscles and joint may remain sore and tender. In these cases it is best to postpone games such as football, in which the player may jar the shoulder frequently, until all the tenderness has passed off.

6. *Traumatic capsulitis* 7. *Periarthritis*

Both these conditions are exercising the minds of orthopaedic surgeons as well as doctors of physical medicine and rheumatologists throughout the world. A tremendous amount of work must be done before the many problems are solved.

These two conditions are considered together, because the treatment for established capsulitis with marked limitation of movement ('frozen shoulder') from either cause is almost the same. Preventive treatment in the early stages may abort this complication and stop the otherwise inevitable fibrosis and adhesion formation. The well established frozen shoulder is a good example of the result of a pathological process ending in fibrosis, either from trauma or degeneration. The young athlete may suffer from it through an inactive indefinite approach to treatment and the older athlete because of the onset of unsuspected degenerative processes.

The traumatised shoulder should recover, by a combination of judicious daily active treatment and careful supervision, without developing into severe capsulitis.

During the early period after the injury, the pain is alleviated by suitable analgesics. This is the negative or non-progressive phase previously described. At this time, particularly, the patient should not work and, if possible, should rest in bed, ideally with physiotherapy brought to his home. A week or two given to rest and treatment at this period may save weeks or months of disability later.

The arm should be supported in a sling, being taken out only twice a day—once for hot sponging and once for contrast bathing with gentle assisted exercises.

As so many thousands of patients suffer from a 'traumatic shoulder' each year it may not be possible to take them off their work, rest them in bed and

FIG. 11. Manipulation of shoulder. Stretching the postero-lateral part of the capsule by drawing the arm across the chest to the opposite shoulder.

provide physiotherapy in their homes, the next best thing is to rest the arm in a universal sling, modify all work as much as possible and arrange easy access to physiotherapy. The patient is given exact instructions on home

treatment—how he can alleviate some of the pain by sleeping on four pillows in the shape of an inverted 'A' with a fifth pillow supporting the elbow, the arm being abducted slightly from the side.

In severe cases, in spite of the greatest care, a degree of traumatic capsulitis develops: and even though gentle manipulations are performed as part of the physical treatment, movements become restricted. Provided there is no bony injury or calcification, a controlled manipulation under pentothal may hasten recovery after eight weeks. This procedure should conform with that described later for the developed capsulitis with adhesions (frozen shoulder).

Differential diagnosis

Periarthritis of the shoulder, unless of traumatic origin is due to degeneration. The degenerative process may affect the paratenon of the rotator cuff muscles and the long head of the biceps brachii, as well as the synovia of the subacromial bursa. The result of this is a plastic exudation with the formation of adhesions which bind down the head of the humerus and the capsular attachments.

We pointed out that, in abduction, the head of the humerus slips and glides into the glenoid fossa; but where adhesions are formed, as in periarthritis the head can no longer glide into its position. When he attempts abduction, the patient simply shrugs the shoulder. It is as if a pot of glue were poured over the head of the humerus and the surrounding structures made to adhere. Neviaser has shown that the capsule thickens and sticks to the humeral head in both the traumatic and degenerative lesions.

In some cases early periarthritis starts acutely, even as a sub-acromial bursitis or stenosing tenovaginitis of the long head of the biceps brachii. It is at this stage, as in the early stages of the traumatic case, that so much can be done to abort the degenerative process. It is imperative to rest the patient in bed, supporting the elbow on a pillow, giving strong analgesics for the pain, eliminating any possible source of infection or gouty deposits and employing the gentlest physiotherapy. Such measures at this early stage may sound extreme and unjustified: but in fact they have served to abort periarthritis in a large number of patients, within a week to ten days—in some cases much earlier, in two to three days.

Occasionally the pain is so great that deep X-ray therapy is justified.

If these drastic measures are not carried out, the condition usually progresses to established capsulitis and 'frozen shoulder'. Whatever the aetiology, the symptoms and physical signs are frequently the same. The patient realizes that movements are becoming restricted because he has pain on certain move-

FIG. 12. Mobilizing the scapula on the humerus: the humerus is held fixed while the scapula is moved in all directions between the manipulator's hands.

ments such as reaching out for the telephone or turning the door handle of a car. He may blame the movement for his symptoms: but the real reason for the pain is that adhesions are forming and movements are becoming restricted; the extra movement makes him aware of his disability. He is now developing 'frozen shoulder'. (Neviaser, 1945).[17]

'*Frozen shoulder*'

The pain varies considerably. (Simmonds, 1949).[18] Some appear to freeze without much pain, others are painful only at night when the patient rolls over on the affected shoulder. In other cases the patient is awakened at a

particular time during the night and the shoulder pain becomes a good alarm clock, waking him at a set time early each morning.

But the pain may be excruciating, particularly in the night. As long as the shoulder is kept still there is no pain, but the slightest movement causes severe pain which may radiate to various parts of the forearm and hand. It may be localized to the thumb and index region or one of the fingers, according to the site of the inflammation and the adhesions in the shoulder. Sometimes, especially in the older patient, the hand becomes involved in the pathological changes, and where this occurs it is always difficult to make the patient symptom free. The hand and fingers become stiff, swollen and oedematous. There may be an associated tenovaginitis of the flexor tendons of the fingers, Dupuyten's contracture, general rarefaction of the bones of the whole extremity and even symptoms related to compression of the median nerve in the carpal tunnel. These cases have often been improperly treated in the early stages.

Vasomotor disturbances, the nosifactor impulses of Sir Thomas Lewis or even loss of muscle tone are possible explanations of this phenomenon: but once oedema of the hand is established it is difficult to break the vicious circle.

The treatment for well-established capsulitis of the shoulder is a lengthy affair. A large proportion of patients have some limitation of external and internal rotation. In others the only limitations are of abduction and horizontal extension; in many all these movements are restricted to some degree. In the majority of patients who have limitation of external rotation, the long head of the biceps brachii is stuck in the bicipital groove, as if involved in a process of stenosing tenovaginitis. Hitchcock and Bechtol[19] have described operations to free the tendon and to remove the intracapsular portion. This treatment may produce quick recovery, in the same way that a similar operation brings quick results in stenosing tenovaginitis in other situations.

The general principles of treatment are best illustrated by reference to a specific instance: for example, a shoulder in which movement is so limited that the patient cannot get his arm into his trouser pocket or do up his braces: in other words he has no external rotation at all—indeed the arm may be fixed in slight internal rotation with only a few degrees of abduction. There is considerable pain, particularly at night and on the slightest movement; the pain radiates to the hand which tends to swell; neck movements are limited in extremes. Radiographs show no abnormality of the shoulder but moderate cervical spondylosis, affecting particularly C5, C6 and C7.

If the process had started with trauma, to reach this stage would take three months. Equally if the onset had been insidious, the history probably goes back several months.

The first object should be to stop the pain. This means putting the arm at absolute rest, except that it is taken out for gentle remedial exercises twice a day after hot sponging, or contrast sponging and physical treatment. The patient is best rested in bed, but if that is impossible, he must wear a universal sling and rest as much as possible. It is imperative for him to stop his work and give himself up to rest and treatment.

Some surgeons believe that only short wave diathermy should be given at this stage, and it is true that many of these patients cannot tolerate other physiotherapy, particularly massage, faradism and manipulations which tend to aggravate the symptoms. Yet if the very gentlest physiotherapy is tried, the pain usually subsides; and provided the patient is rested, the movements start to free. As soon as the treatment appears to be beneficial, gentle daily manipulations may be added to the physiotherapy.

From the beginning hydrocortisone injections are given twice a week. If external rotation is limited, the main object should be to free this movement: therefore the injections should be given into the long head of the biceps brachii. If the surgeon concentrates on this tendon the movement may show improvement in a week to ten days. So long as the movements are progressing satisfactorily, one should never attempt forcible manipulation. On the other hand, controlled manipulation under general anaesthesia should never be considered until the patient has improved, the improvement being evidence that the negative phase has stopped and that the positive phase of healing has commenced. Thus the time for manipulation is when the shoulder, after initial improvement, has reached a stationary phase: but it is quite contra-indicated as long as there is pain at night, for it may start a reaction which aggravates the symptoms and reverses the improvement in the range of movement.

Sometimes, in spite of all efforts, the shoulder continues to freeze. An attempt should be made to stop the process, either by giving a course of deep X-ray therapy or by injection into the stellate ganglion. Either of these procedures may alleviate pain and check the degenerative process. A sign to look for is fibrillation or fasciculation of the muscles, with stuttering on faradic stimulation. This is evidence suggestive of three possibilities: the condition is still in the negative phase: there is a septic focus somewhere: or there is an associated motor neuron disease or even a cerebro-vascular disturbance.

To return to the sort of case under discussion—of one seen for the first time after a history of three months gradually increasing disability, pain usually stops after a week's treatment and the movements begin to improve. At the end of three weeks a controlled manipulation under a general anaesthetic may then hasten recovery. We have already stated the criterion for manipula-

tion—that there should be no pain at night and external rotation, after initial progress, should have stopped or almost stopped movements improving. The manipulation should concentrate on external rotation alone. It is unwise to attempt to improve abduction as well.

The shoulder is manipulated by traction and counter traction; the involuntary movements are carried out, the head of the humerus being pushed down into the glenoid, forwards into its socket and backwards into its socket. The capsule is stretched in all directions, including horizontal adduction, and then the full range of voluntary movement is carried out. Finally, the cervical spine and the elbows are manipulated. If external rotation is limited, this movement only is increased by manipulation at this stage. It is far wiser to let the patient recover, giving him three or four weeks further gentle treatment and then to do another manipulation to obtain abduction. By these methods, a markedly frozen shoulder is often well on the way to recovery in six to eight weeks.

On the average, the shoulder with a traumatic aetiology recovers in three months: the shoulder with the history of insidious onset, associated with a degenerative process, three and a half months. After the manipulation under full general anaesthetic, the manipulator must be prepared to see the patient for periods of at least a quarter of an hour daily for the first ten days. The physiotherapist is the first to see the patient each day, giving very cautious gentle treatment and encouraging active assisted movements. Treatment at this stage consists of massage, faradism and shortwave with addition of further injections of hydrocortisone at weekly intervals.

When the patient has obtained a satisfactory result, he must be warned that he will have to continue home treatment indefinitely, otherwise the symptoms are liable to recur. Also he should report any symptom in the opposite shoulder since, as a result of the extra strain on it, a capsulitis is often induced. He must massage his cervical spinal muscles after contrast baths and hot baths, and perform gentle exercises, not only for the shoulder joint, but also for the shoulder girdle and cervical spine.

To reiterate and summarize: capsulitis of the shoulder joint, whether due to injury or insidious degeneration, may be aborted by appropriate means, of which rest and very gentle physiotherapy are the most important. In a well-established case pain must be stopped by rest, appropriate physiotherapy and hydrocortisone injections. No forcible movements or manipulations are given until all pain has stopped, especially at night, and the patient is beginning to show improvement. If there is limitation of external rotation, this movement must be obtained first and it is better in some cases to do a series of manipulations under anaesthesia to obtain external rotation and then abduc-

tion, rather than to try to get all the movements in one manipulation. At each session the neck and elbow are also manipulated. It is by a combination of rest from all forms of strain and anxiety, gentle remedial exercises, physiotherapy, hydrocortisone injections, deep X-ray therapy, manipulations, analgesics, and an active, sympathetic approach with constant encouragement that the capsulitis of the shoulder joint is made to recover in as short a time as possible.

INJURIES TO THE CLAVICLE

1. *Acromio-clavicular joint*

Falls on the shoulder joint or the point of the collar bone are notorious for causing subluxations of the acromio-clavicular joint. The subluxation may be momentary, the outer end of the clavicle springing back into position. After the recoil there may be only a slight reaction. If the injury is more than slight, the subluxation may cause tearing of the capsule and even rupture of the conoid and trapezoid ligaments. In the latter event the joint is disrupted and dislocated with marked prominence of the outer end of the clavicle which may even stick out under the skin, but it is more common for the capsule to be moderately distended and the ligaments to be torn so that subluxation remains after the injury. There may be:—(a) sprain of the joint, (b) subluxation of the joint, (c) dislocation.

In subluxation Horn[20] describes tears of the trapezius and deltoid muscles as well as rupture of the conoid and trapezoid ligament. A fragment of the articular cartilage may become loose inside the joint. Apart from the effect of the subluxation, there is often contusion and bruising of the tissues of the shoulder joint and the outer end of the acromion. The shoulder joint itself may suffer any of the injuries already described. Whatever the degree of injury, active treatment is essential.

Marshall Urist[21] considers that from a multitude of methods of treating acromio-clavicular subluxation, only two are worthy of consideration in the athlete. The first is his method of immobilizing the elbow and chest together with plaster of Paris bandages down to the iliac crest, and then fixing the reduced acromio-clavicular joint by means of a felt pad and a strip of rubber taken from the inner tube of an automobile tyre to the elbow and chest plaster cast. This is maintained for five weeks. If the acromio-clavicular joint remains consistently painful he advocates removal of the outer $\frac{3}{4}$ in. of the clavicle. Both these methods are excellent, but a third is suitable for recent cases. Hydrocortisone, procaine or xylocaine with hyaluronidase is injected immediately. Active physiotherapy and home treatment are started at once.

On the third or fourth day serum will often have seeped to the surface and may be aspirated through a large bore needle. For protection and support water-proof elastoplast is applied over a felt pad and the point of the elbow is elevated by a universal sling. The patient is encouraged to remove the sling to carry out gentle shoulder joint movements, in order to prevent the development of traumatic capsulitis. Home treatment consisting of hot sponging, contrast baths and massage can be given with the strapping in place; for physiotherapy consisting of massage, faradism and short wave, only the universal sling need be removed. By this active approach most cases are symptom free in a fortnight and with a further fortnight's rehabilitation can stand up to vigorous games such as rugby football. Some degree of subluxation usually persists. When the athlete starts playing his sport again, the joint should be supported and protected with a felt pad and strapping.

Severe subluxation or complete dislocation should be operated on immediately, so that any sequestration of the articular cartilage can be removed at operation. The torn ligaments are reconstructed and reduction of the dislocation maintained by three Austin Moore nails passed transversely inwards through the outer end of the acromion, so as to sit on the top of the outer end of the clavicle. The nails are maintained in position for eight weeks.

A perfectly satisfactory result can be obtained in chronic cases by removal of the outer 1 in.-1½ in. of the clavicle. This operation has even been performed on both sides. The patient has subsequently been able to carry out vigorous and violent exercises. He may even play rugby football. Complications to avoid are:—

1. A tender, painful acromio-clavicular joint
2. Traumatic capsulitis of the shoulder joint
3. Tender thickenings in the surrounding muscles, such as the trapezius deltoid and pectoral muscles
4. Involvement of the cervical and shoulder girdle muscles leading to brachial neuritis.

2. Sterno-Clavicular subluxations

These occasionally occur as the result of a direct blow on the back and outer side of the shoulder so that the direction of the force of the injury is from without inwards along the clavicle to its inner side. Active treatment to dissipate the products of bruising and contusion should be started immediately with pressure pad and strapping support. Occasionally a chronic painful subluxation requires operative intervention either to reconstruct the joint or to remove the inner 1½ in. of the clavicle.

REFERENCES

1. Watson Jones, R. (1952-5.) *Fractures and joint injuries.* 4th edn.
2. Mennell, James B. (1949.) *The Science & Art of Joint Manipulation.* J. & A. Churchill, Ltd., London.
3. Nissen. K. I. (1959.) Pain in the arm and the carpal tunnel syndrome. *Postgraduate Medical Journal*, London, July, p. 379.
4. Furlong, R. (1953.) *Practitioner*, September, p. 320.
5. Garden, R. S. (1961.)Tennis elbow. *J. Bone Jt. Surg.*, Vol. 43B, No 1, p. 100.
6. Trethowan, W. H. (1929.) *Brit. J. Surg.* 2:1218.
7. de Góes, H. (1960). The radio-humeral 'meniscus' and its relation with tennis elbow. *Archives of Interamerican Rheumatology*, Vol. III, No. 4, December, 1960.
8. Pridie, K. H. Personal communication.
9. Mennell, J. B. (1947.) *Physical Treatment by Movement, Manipulation and Massage.* J. & A. Churchill, p. 189.
10. Miller, J. E. (1960). Javelin thrower's elbow. *J. Bone Jt. Surg.*, Vol. 42B, No. 4, p. 788, November.
11. Seddon, H. J. (1949.) War injuries of peripheral nerves. *Brit. Journal of Surgery.* War Surgery Supplement, No. 2, p. 325.
12. Saha, A. K. (1958.) Zero position of the gleno-humeral joint: its recognition and clinical importance. *Annals R. Coll. Surgeons*, Vol. 22, No. 4, April, p. 223-236.
13. Ellis, V. H. (1949.) Painful shoulder: discussion at Brit. Orth. Assn. Meeting, Belfast. *J. Bone Jt. Surg.*, Vol. 31B, p. 132, February.
14. Ellis, V. H. (1949.) *Ibid.*
15. Hitchcock, H. H. and Bechtol, C. O. (1948.) *J.B.J.S.*, Vol. 30A, No. 2, April, p. 263.
16. Hitchock, H. H. and Bechtol, C. O. (1948.) *Ibid.*
17. Neviaser, J. S. (1945.) Adhesive capsulitis of the shoulder. *J. Bone Jt. Surg.*, Vol. 28A, No. 2, April, p. 211.
18. Simmonds, F. A. (1949.) Shoulder pain with particular reference to the 'frozen' shoulder. *J.B.J.S. Brit.*, Vol. 31B, No. 3, August, p. 426.
19. Hitchock, H. H. and Bechtol, C. O. (1948). *Ibid.*
20. Horn, J. S. (1954.) *J. Bone Jt. Surg.* The traumatic anatomy and treatment of acute acromio-clavicular dislocation, p. 194, Vol. 36B, No. 2, May.
21 Urist, Marshall R. (1959.) The treatment of dislocations of the acromio-clavicular joint. A survey of the past decade. *Amer. Jl. of Surgery*, Sept. A Symposium on Sports Injuries.

6

HEAD, NECK AND TRUNK

Contusions of the scalp

The scalp consists of five layers and naturally treatment will depend on the extent of the injury and which layer has suffered most severely. They are:— skin, connective tissue, aponeurosis of the occipito-frontalis or epicranium muscle, loose connective tissue, pericranium or periosteum. Active treatment is initiated immediately with consequent good result. Some of these injuries are associated with compound wounds which must receive, without hesitation, the full treatment accorded to such wounds.

The cauliflower ear of the boxer or rugby football forward is a typical example of allowing a clot to remain in situ, organise and give rise to an enlarged thickened painful ear. In a recent case active treatment including expression of haematoma, aspiration or the employment of local anaesthetic and hyaluronidase allowed for absorption and sound healing within ten days. In these cases if possible the patient should wait a further ten days before running the risk of a recurrence and the ear should be protected by a suitable skull cap.

Concussion injuries from knock-outs

The pathology of these injuries has been worked out extensively by Rowbottam[1] and Northfield[2] and discussed in a Symposium of Sports Injuries (1959)[3] and in O'Donoghue's book Injuries to Athletes.[4] A blow on the chin causes the condyle of the mandible at the tempero-mandibular joint to be forced upwards against the under surface of the temporal bone. The repercussions cause contusion of the base of the brain above this with the occurrence of small haemorrhage with loss of consciousness for a variable length of time. Repeated blows with disturbance of the normal brain equilibrium produces in time a condition of punch drunkness.

Lesions of the tempero-mandibular joint

Traumatic: The tempero-mandibular joint has an intra-articular cartilage or meniscus which is prone to contusions, bruising or tears and splits in the same way as the menisci of the knee. Boxers and footballers particularly are liable to sustain injuries to this joint. Often in the first few days there is in-

ability to approximate the teeth and eating is extremely difficult. Provided the meniscus is not fractured active treatment provides a symptom free result in three weeks. Occasionally removal of the meniscus proves necessary owing to loud clicking or recurrent locking.

Contusions of the thoracic wall

These are interesting injuries as they demonstrate so well the various aspects of the tissue bruising and contusion. Treated actively symptoms clear up even in severe cases in three to four weeks. Left untreated or allowed to rest completely symptoms often persist for years.

The interlacing arrangement of the intercostal muscles covered as they are by a tough fascial layer, tends to limit the spread of traumatic effusion, so that at one phase in treatment it necessarily must be vigorous, even if the superficial tissues become more tender or the deep surface of the muscles and the pleura become involved with sometimes increase of pain. The patient requires to be reassured. The seeping process of the products of traumatic

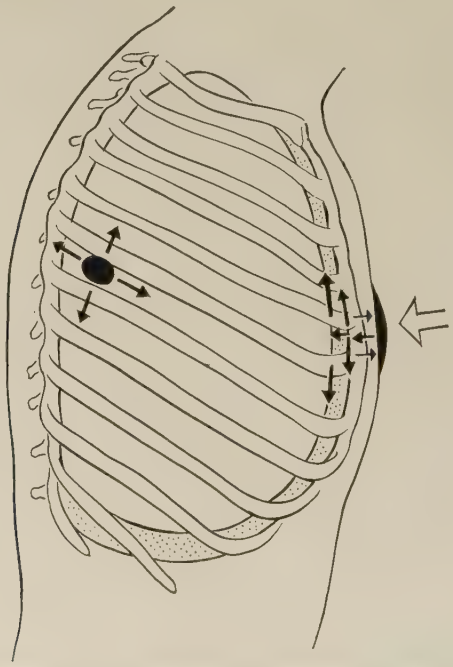

FIG. 1. Showing effect of contusion of chest wall: how the spread of traumatic swelling is forwards, backwards, downwards and upwards in an intercostal space.

effusion is true to form and spreads forwards, backwards, outwards, inwards, laterally and medially so that a contusion in one of the intercostal spaces becomes superficial by spreading outwards along the intercostal space but is limited above and below by the adjacent ribs. The spread being also backwards in a few days the surface of the pleura becomes involved and is heralded by pain on breathing and the presence of a pleural rub. These contusions of the intercostal muscles naturally may be complicated by involvement of overlying muscles such as the pectorals, serrati, rhomboids and latissimus dorsi and contusion of the ribs even to fracture. Active physiotherapy must include short wave diathermy and faradism provided there is no fracture. Protection and support is essential and tends to give great relief as soon as applied. Preferably felt and webbing are ideal but a removable many tail binder, coarse towelling binder or corselet are also satisfactory and in fact the latter which allows for little disturbance is the best.

FIG. 2. Technique to mobilise the upper cervical joints. The operator's left hand transmits the thrust through the index finger.

A word must be said here against support by strapping or elastoplast. In associated fractures some physicians are against the use of support because it tends to restrict full respiratory excursion on the unaffected side. The use, however, of a corselet or felt and webbing support gives such instant relief

FIG. 3. Manipulation of the lower cervical and cervico-dorsal junction. The manipulator's left hand acts as a fixed point while the right hand moves the apophyseal joints above this fulcrum.

that respiration generally becomes immediately more normal. Strapping or elastoplast unless over stockinette or a bandage tend to promote skin blisters and abrasions and do not allow for appropriate physiotherapy or home treatment.

Professional steeplechase jockeys who probably, more than any other band of athletes, sustain injuries to the thoracic wall, favour felt and webbing or a corselet as the support can be easily removed for bathing, home treatment and regular physiotherapy.

It is criminal to apply strapping or elastoplast directly on the skin and in every outpatient department there should be written up clearly a warning to the young house surgeon against committing this atrocity in treating injuries of the chest wall.

Strains

Strains of the intercostal muscles as well as the pectorals, serrati, trapezii, levator angulae scapulae, rhomboids and latissimus dorsi produce similar symptoms to those of contusion.

FIG. 4 a and b. Upper dorsal technique. The patient's right arm is firmly fixed across her chest so that the manipulator's thrust will not be dissipated in distracting the scapula from the chest wall. The left hand is used to localise the movement to a selected area.

Frequently strains of the serrations of the intercostals, latissimus dorsi and external obliques will occur together and cause severe symptoms. Such strains are common in fast bowlers and will occur on the left side in the right-handed bowler. With injection therapy, active physiotherapy and home treatment, there is usually complete recovery with return to full bowling in ten days. Left to a negative inactive approach to treatment many cases are seen with symptoms even months later.

FIG. 5. Technique to mobilise the lower dorsal spine. The thrust is transmitted through the manipulator's right hand. The patient's forearm should be at right angles to the plane of the apophyseal joints.

Contusions of the posterior spinal muscles

These cases are often combined with fractures of the transverse processes and sometimes an enormous amount of traumatic effusion. The following case illustrates this point. A polo player sustained a severe injury of this type when his horse fell and rolled on him. The pommel of the saddle hit him in the perineum and he was kicked by the horse in the loin. He bruised and contused the urethra and had to have a catheter passed. A pyelogram revealed extensive contusion of the renal pelvis on the left side with marked displacement of the left kidney. There was present an enormous distension of the issues of the left side of the dorso-lumbo-sacral regions. There was no frac-

ture but the swelling fluctuated and appeared to consist of two separte compartments, a high one above the twelfth rib, and a lower one below the twelfth rib. Operative removal of the effusion was arranged and carried out two days after the injury through two T-shaped incisions 1½ in. in length. About two litres of blood and serum was removed in the first instance and over the next six weeks decreasing quantities of blood and serum seeped through. It was estimated that over six litres of fluid substance were withdrawn before the wound dried up completely in five weeks. The patient was practising at polo after four weeks in spite of the fact that the wounds were still being tapped each day and he was still having physiotherapy.

FIG. 6. Manipulation of the lumbar spine. The thrust is made with the manipulator's left elbow and the counter thrust with the right hand against the patient's shoulder. The fingers of the left hand are free to feel movement between the lumbar vertebrae.

Extensive bruising and contusion frequently accompany cracks in the sacrum or injuries to the coccyx. Active treatment makes even the severe ones symptom free within six weeks whereas with an inactive approach the case will become chronic and give rise to pain and disability for months or even years. This particularly applies to contusion sprains of the various coccygeal joints, expecially the sacro-coccygeal. Persistent pain in these cases is blamed

on to lesions of the lumbo-sacral discs. With active treatment spread of effusion seeps into all the surrounding tissues including the front of the sacrum and coccyx as well as towards the lumbo-sacral articulations. This may account for the occurrence of low back pain associated with tender points connected with the posterior aspect of the sacrum and coccyx. It is possible also for the original injury to be the cause of a concomitant disc lesion.

FIG. 7. With the heel of the manipulator's left hand against the ischial tuberosity the ileum is lifted and rotated from the sacrum.

Muscle strains or tears of the posterior spinal muscles

The treatment of these follows the normal plan of management of muscle injuries in general. Sometimes extensive swelling occurs with haematoma formation. Dispersal of the traumatic swelling and reconditioning of the muscles are carried out by the active therapeutic approach. If an athlete is suffering from restriction of range of movement more than eight weeks after injury, a manipulation under general anaesthesia may be indicated.

REFERENCES

1. Rowbottom, G. F. (1949.) *Acute Injuries of the Head*. E. & S. Livingstone, Edinburgh.

2. Northfield, D. W. C. (1953.) Head injuries: brain injury and its diagnosis. *Brit. med. J.*, January 10, p. 90.
3. Murphey, F. and Simmons, J. C. H. (1959). A Symposium of Sport Injuries. *Amer. Jl. of Surg.*, September, p. 379.
4. Wilkins, H. (1962). Craniocerebral Injuries in Athletes from D. O'Donoghue's book *Injuries to Athletes*, W. B. Saunders Co., Philadelphia and London, p. 77, Part III, Chap. 5.

7

LOWER LIMBS

Contusions in the lower extremity

Injuries of this sort in the lower limb may be considered together since, whatever their situation, they have much in common. Except for difference of detail, the principles of treatment are the general ones discussed on page 320.

Contusions over bony points will be considered first, then those which involve muscles. Obviously there is sometimes a combination of both.

Contusions over bony points

Common sites for contusions are:—

(a) the greater trochanter
(b) the ischial tuberosity
(c) the patella
(d) the head of the fibula
(e) the subcutaneous border of the tibia
(f) both malleoli
(g) the dorsum of the foot
(h) the phalanges

As there is usually swelling and tenderness of the structures in the vicinity of the point of injury, it is wise to have radiographs to exclude a fracture. Chip fractures at any of these sites or stellate fractures of the patella are common as the result of falls, kicks, knocks, or blows: and the amount of reaction varies with the size of the blood vessels involved.

The main object of treatment is to promote quick absorption of the resulting traumatic effusion in order to prevent the formation of thickenings which are liable to remain tender and painful for months. Traumatic effusion may spread some distance from the original site of injury, manifesting itself as remote bruising: it must not be allowed to lodge in the vicinity of vital structures, the function of which it may affect. Active treatment follows the lines set out in Part III, Section I, Chapter 4.

Each site of injury has some special problems:—

(a) *the great trochanter.* A haematoma spreads down the iliotibial band to-

wards the bursae about the insertion of the tensor fascia femoris and the glutei into the greater trochanter. There is a tendency for the knee flexion to be lost, and the patient must guard against this by performing gentle flexion exercises especially after a hot bath. If the bursae become involved certain specific movements will cause pain. Treatment must be directed to overcoming this complication as soon as possible.

(b) *the ischial tuberosity*. Apart from actual contusion of the bone the superimposed bursa is bound to suffer injury. Traumatic effusion tends to spread down the hamstring muscles from their origin.

(c) *the patella*. The superficial surface of the patella and the overlying prepatellar bursa are very vulnerable. The under articular surface may suffer a contra-coup injury which is sometimes followed by chondromalacia patellae. The effusion spreads in the infrapatellar pad of fat and there may be concomitant synovitis of the knee joint. It may be necessary to continue treatment for several weeks until there is no chance of permanent disability from any of these complications.

(d) *the head and neck of the fibula*. The lateral popliteal nerve is so close to the bone that one must always suspect nerve injury. If there is any doubt, it is well to consider a light removable plaster of Paris splint which may be put on at night to keep the ankle at a right angle. If the injury is extensive there may conceivably be an associated anterior tibial compartment syndrome, which might require extensive decompression of the tissues.

(e) *the subcutaneous border of the tibia*. Proper evacuation of the haematoma is essential or a thickened painful mass of fatty tissue may form under the skin. The incision must be kept open and, of course, sterile. A spreading haematoma and traumatic effusion here can simulate a septic wound or an area of thrombo-phlebitis. Tender, painful, persistent thickenings of the subcutaneous border of the tibia can be avoided only by adequate treatment, which may have to continue for several weeks.

(f) *both malleoli*. Tender painful thickenings are also liable to form here unless treatment is adequate. Adventitious bursae may form as a result of the pressure of shoes or boots.

(g) *the dorsum of the foot*. Direct injury may be associated with fractures of the tarsal or metatarsal bones. If the swelling is large and diffuse, multiple small incisions down through the deep fascia may be necessary. Active treatment with frequent baths and exercises must be instituted, great care being taken to keep the small wounds sterile.

(h) *the phalanges*. If a haematoma is distending the nail bed it should be punctured and the haematoma allowed to escape. This will relieve tension and pain immediately. The toe must be protected and supported by suitable

dressings and the patient encouraged to wear a proper shoe. By these methods cricketers have been able to return to the game within a week even with a fracture.

Contusion of muscles

The muscles most liable to contusion are those of the buttock, thigh, anterior tibial and calf. The treatment is similar to that for contusion involving bone. The incision through the skin and deep fascia may not at first allow traumatic effusion fluid to escape, but after exercises and faradic stimulation of the muscles, blood and serum will pour out, especially if the wound is kept open each day.

(a) *contusion of the gluteal muscles.* There is a tendency for the products of traumatic effusion to spread downwards, especially to the bursae round the greater trochanter. Skiers are particularly prone to these injuries. In some cases, serum seeps out of the muscles and forms a boggy swelling which can be easily tapped by a large bore needle. The amount of traumatic swelling is enormous in some cases and the patient has to undergo daily treatment. If a modified incision is made and if home treatment and daily treatment are continued the patient can often carry on with his occupation; but he should avoid strenuous exercise.

(b) *contusion of the quadriceps.* This often leads to myositis ossificans especially when there is a haematoma deep in the substance of the muscle.

If, as is often the case, the thigh, knee and calf are completely immobilised in a plaster cast, the knee joint will become stiff and there will be complete loss of flexion. This may take months to return and may never be full. Recently a professional footballer was examined to assess the end results of a defined myositis ossificans on the lateral aspect of the middle third of the femur. It had kept him out of football for eighteen months.

If contusion in the quadriceps is treated properly from the start, however, the prognosis is quite different. This is illustrated by an amateur footballer seen within a week of injury. He could not afford to be off work, so a small incision was made over the point of maximum swelling and tenderness. He was given a removable plaster of Paris cast which prevented him flexing the knee but allowed him to bear weight on the leg. He was treated actively with injection therapy and massage to the knee and the structures above but no massage was given to the area of swelling. He performed active flexion movements of the knee, but only when he was not bearing weight on the leg. Although a limited amount of myositis ossificans formed, he was completely symptom free in three months with full flexion of the knee.

It is often a problem to decide exactly the right procedure for those cases seen between the end of the third and eighth week in which there is an established myositis ossificans with almost complete loss of knee flexion. There is no doubt that this loss is due to two causes:—

(1) the binding and matting down of the origin of the quadriceps by the organisation of the haematoma into fibrous tissue and bone.

(2) seeping of the haematoma and its products to the structures around the knee, including the suprapatellar pouch and expansion of the quadriceps.

These conditions lead to loss of the extensibility of the quadriceps and this limits flexion of the knee.

The following is a line of treatment which produces a reasonably quick result in the established case. The patient's leg is rested in a removable plaster of Paris cast so that he cannot flex the knee when he is bearing weight. Injections of procaine 2 per cent 5 ml. and 1000 units of hyaluronidase are given at weekly intervals. Active flexion exercises (without weight bearing) are started immediately but some resistance to the movement is provided by a pillow over the edge of the bed. These are in fact strong isometric contractions of the hamstrings, and they cause reflex relaxation of the quadriceps. Massage is given avoiding the area of contusion in the muscle, and also gentle faradic stimulation to the quadriceps. The knee is manipulated very gently with the patient prone. The surgeon must use no force, for his object is only to free capsular adhesions around the knee joint itself. By these methods it is possible to limit the amount of myositis ossificans to an absolute minimum, and to restore the function of the knee joint in as short a time as possible. Those patients who do develop gross calcification should be given a period of complete rest in plaster for six weeks before active treatment begins. Then flexion movements should be carefully performed by active contractions of the hamstrings. No forcible manipulative movements should be applied or muscle fibres attached to the area of calcification may be torn with further haemorrhage and further calcification.

Occasionally callus formation obstructs the action of the quadriceps muscles. When the process is complete, it may be necessary to remove the excess bone by operation.

(c) *contusion occurring in the anterior tibial group of muscles.* Large effusions may be produced by relatively trivial blows. The effect may be to cause the 'anterior tibial compartment syndrome', with evidence of involvement of the peroneal nerve (lateral popliteal) and even necrosis of the muscles of the compartment. If the surgeon has the slightest evidence of such a develop-

ment, he should operate at once, splitting up the fascial covering, thus decompressing it and relieving tension. Less severe contusions may be treated by the active conservative methods already described; but treatment may have to continue for some weeks to avoid tender thickenings in the tissues, especially along the attachments of the muscles to the tibia and fibula.

(*d*) *contusion of the calf muscles.* Unless treated actively these are often liable to lead to shortening of the tendon Achilles with pain on walking and inability to run. Bruising spreads downwards around the ankle and foot and must be dispersed by active treatment. Modified walking can often be allowed provided the heel is raised by a sorbo rubber pad and the ankle and leg supported by an elastic bandage. A walking stick may help the patient to get around.

Muscle strains

(a) *strains of the buttock muscles.* Pain and tenderness of the gluteal muscles, especially of the gluteus medius, is so often associated with a lower lumbar disc episode that the doctor often forgets that primary strains of these muscles are not uncommon.

Postural and occupational strains may occur without a tear even of a few fibres of these muscles, particularly when a patient whose posture is habitually poor makes excessive demands on them—a bowler, for example, at the beginning of the cricket season. These muscles are prime fixors of the pelvis to the thigh at the hip joint, and through excessive use may become strained and tender and lose their terminal extensibility.

Such a patient should be taught proper posture and given postural training exercises, physiotherapy, home treatment, support and then return to athletic activity gradually.

(b) *strain of the iliopsoas muscle.* This gives rise to tenderness anteriorly just in front of the hip joint ; the patient may have difficulty in flexing the joint, especially against resistance, and the movement is usually painful. It is often associated with inflammation of the psoas bursa in someone prone to rheumatism. Extension of the hip joint may be limited and painful if forced. The ordinary routine treatment—rest from strain, graduated exercises, physiotherapy, home treatment and local support may produce a cure, but these cases are often resistant to treatment. One should search for an obvious septic focus and evidence of gout. Resistant cases sometimes respond to a course of deep X-ray and moderate doses of butazolidin. Support, in the form of an elastic webbing bandage is often helpful when the patient starts gradually to use the joint.

(c) *strains of the thigh muscles.* Tears of the rectus femoris at its point of junction with the main part of the vastus intermedius are fairly common. The retracted torn fibres of the muscle form a tender bulge in the middle third of the thigh. Sometimes the tear is at a higher level, where the reflected and straight heads join. Active treatment, including injection therapy, can make these cases symptom free in ten days. It is wise to insist on a further ten days of graduated activity before the patient takes part in violent sport.

(d) *strains of the quadriceps apparatus.* These are notoriously slow to resolve. If there is a tear of any magnitude it should be repaired by operation, especially if the patient is young. Moderately good results can be obtained in the older athlete by active treatment consisting of a removable support preventing flexion of the knee when weight bearing, but which can be removed for non-weight bearing flexion exercises, massage and faradism. It may be possible to aspirate effusion from the area of tear or the knee joint itself. However, if possible, more than moderate degree of tearing should be repaird surgically.

(e) *strains of the hamstring muscles.* Slight and moderate tears of the semitendinosus, semimembranosus and biceps femoris muscles are very common and often cause prolonged disability and constitute the typical Charley Horse injury. One or more of these muscles are torn either at their upper attachment, middle or lower attachment. As typical of other muscles injuries, they are liable to occur at the beginning of a game or race before the athlete warms up or during some instance in a match such as during a difference of opinion with another player so that there is loss of concentration, or at the end of a game when muscular fatigue is beginning to occur. Often a tear of the short head of biceps femoris unless complete healing takes place will be followed by a tear of the long head. Full extensibility and power must be established before the athlete can take part again in strenuous sport. So often this type of injury is rested and tried out again in about two weeks with the result that there is a further tear. These injuries must be treated actively with rest from strain but gentle remedial exercises without strain, injection therapy and physiotherapy. If the tear has been more than moderate, extensive bruising may appear down the calf to the ankle and is an indication that active treatment has been instituted.

At the end of three weeks good healing of the torn muscle should have occurred and with carefully graduated exercises it is wise to take a further fortnight to three weeks before attempting to take part in a vigorous game.

If torn hamstrings have been allowed to heal in a contracted shortened

state, manipulation under full anaesthetic may be indicated. It is sometimes wise to carry out this procedure at the end of four weeks before the athlete has returned to vigorous sport, so as to ensure the full extensibility of the torn muscle.

KNEE JOINTS

Gray's Anatomy (1960)[1] points out that at first sight the knee joint appears to be one of the most insecure joints in the body: it is subject to severe lever action, as the bones which make up the joint are the longest in the body; the articular surfaces are not congruous; and the range of movement is great. To make up for these mechanical deficiencies the joint can be controlled by the powerful surrounding muscles and the bones held together by particularly strong ligaments.

Nevertheless in every form of athletic activity, the knee joint placed as it is between the hip and ankle joints, is very vulnerable. It is liable to be damaged by both direct and indirect trauma.

Movements

Galaezzi (1927)[2] described the rotation of the tibia on the femur in flexion and extension.

The lower leg is free to move at the knee joint. During the last few degrees of extension the tibia rotates laterally, 'screwing home' the joint as it were. In the last few degrees of flexion it roates medially on the femur. When the full weight of the body is carried on the leg, however, this rotation is carried out by the femur on the tibia both in flexion and extension. Thus in extension there is medial rotation of the femur on the tibia and in flexion lateral rotation of the femur on the tibia. These points have recently been stressed again by Helfet (1959)[3].

In flexion or extension the tibia also skids or glides backwards and forwards on the femur. Throughout flexion and extension the axis of pivot of the tibia around the lower end of the femur is constantly shifting. In extension the axis is almost midway between the centres of the femoral condyles. In flexion it is placed further back and at a higher level. The patella also changes its relative position as the joint moves. When the knee is flexed at a right angle, the tibial tuberosity points to the mid-line of the patella. In full extension the line of the tibial tuberosity is near the lateral border of the patella. Helfet (1959)[4] states 'When the knee is bent, the patella is carried downwards and backwards on to the under aspect of the femur where the trochlear surface is prolonged on to the medial femoral condyle'. The articular surfaces of the

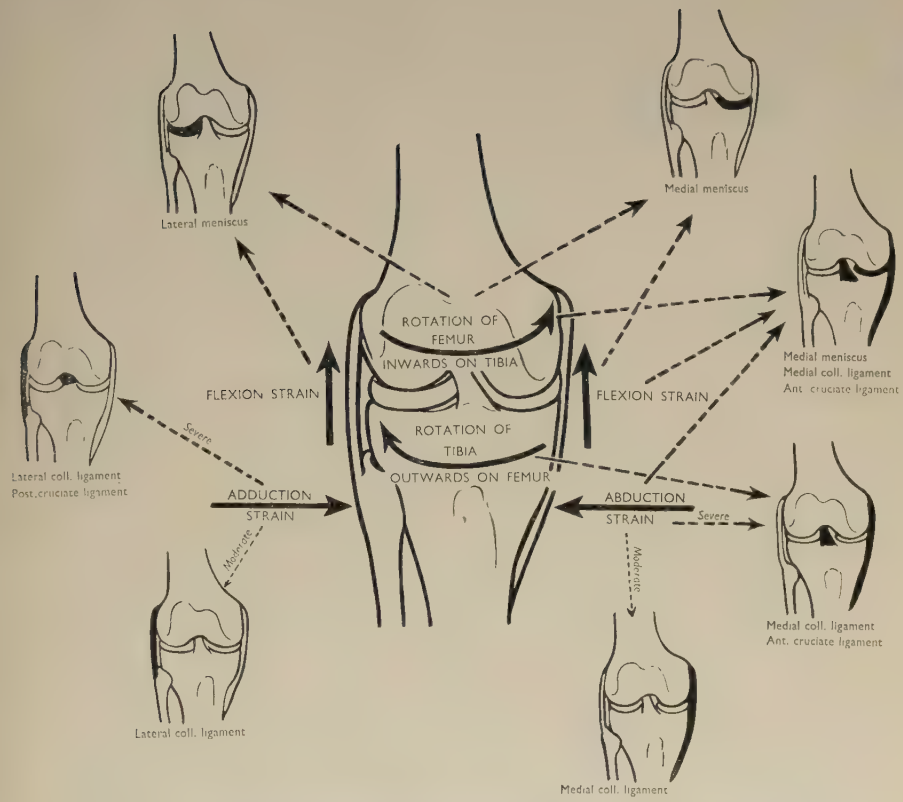

FIG. 1. Different types of injuries caused by one force or a combination of forces.

patella are divided into a large lateral and smaller medial one, and it is the medial surface which is in contact with the trochlear surface of the medial condyle in flexion.

The elaborate relationship which the ligaments and meniscii maintain to one another during the locking and unlocking of the joint, called by Platt and quoted by Helfet, 'figure of eight anatomy', depend for their integrity on perfect timing. So long as the movements are synchronous there is no strain on the attachment of the meniscii or on the ligaments themselves: but when there is an uncontrolled, forced movement whether in extension, flexion, abduction, adduction or a combination of all these movements, the timing is put out and injury is liable to occur. Fig. 1 illustrates these points and suggests the structures which may be injured in various types of strain.

Ligaments

These are natural thickenings of the capsule of the joint on the inner and outer aspects and consist, fundamentally of (a) the medial collateral ligament, make up of a superficial long anterior part and a short deeper part which is attached to the medial meniscus: (b) the lateral collateral ligament which is surrounded at its attachment to the head of the fibula by the insertion of the biceps femoris tendon. It has no attachment to the lateral meniscus: (c) the cruciate ligaments which are vestigial remains of the lateral ligaments of the knee joint.

In the lower animals the joint is divided into two separate compartments. The anterior cruciate ligament stabilises the forward movement of the tibia on the femur. When it is ruptured one can draw the tibia forwards on the femur (anterior drawal test). In some instances the tibia drops backwards in relation to the femur and at first sight there appears to be a rupture of the posterior cruciate ligament. When a definite rupture of this posterior cruciate ligament occurs the tibia can be actually forced backwards on the femur (posterior drawal test).

Various accessory or secondary ligaments have been described and their position mapped in detail. Last[5] considered that some of these accessory ligaments are manufactured by the methods of investigation. Structures which are infiltrated with fluid before dissection often look like ligaments during dissection, but are in fact not true ligaments.

The following ligaments have been described by various anatomists:—

(1) Galeazzi described a strong fibrous band connecting the tibial attachment of the anterior cruciate to the anterior horn of both the medial and lateral meniscii. Secondly, a strong band connecting the posterior horn of the lateral meniscus to the upper femoral attachment of the anterior cruciate ligament.

(2) Winslow has described two ligaments: the transverse ligament of Winslow connecting the front of the medial meniscus to the front of the lateral meniscus: and the oblique popliteal ligament which is a part of the thickening of the posterior capsule centrally.

(3) Barkow has described the ligament connecting the anterior horn of the medial meniscus to the posterior horn of the lateral meniscus.

(4) Kaplan (1961)[6] points out that ligamentous structures are functionally and morphologically connected with the corresponding muscles. He describes, when a lateral fabella is present, a fabello-fibular ligament which takes the place of the short lateral ligament. This latter ligament is in itself an offshoot of the lateral arch of the arcuate ligament. The arcuate ligament covers

the popliteus muscle and extends as a medial and lateral band on both sides of the tendon.

(5) Humphrey and Wrisberg described accessory ligaments passing before and behind the posterior cruciate ligament.

Muscles

The quadriceps muscles, particularly the vastus medialis, play an important part in the 'screwing-home' action of the tibia on the femur during the last few degrees of extension.

Robert Jones, Timbrell Fisher, Smillie and many others have stressed that after injury there is often quadriceps wasting and consequent weakness. As a result the knee tends to give way because the weak vastus medialis is unable to perform the final screwing home which gives stability to the extended joint.

Many of the muscles which control the movement of the knee joint also help to move the hip or ankle. The sartorius, gracilis and semitendinosus for example move the hip as well as the knee; the gastrocnemius, which originates from the posterior aspect of the lower end of the femur, acts both on the knee and the ankle. In injuries to the knee joint, the muscles and tendons around the joint may be affected; so that one may expect derangement of the movements not of the knee alone but of the hip and ankle joints as well.

Prevention of Injury

The medical advisers and trainers of some American college football teams insist on the firm strapping of both ankle and knee joints whether or not these joints have suffered previous injury. They have statistical evidence that this measure diminishes injuries to these two joints. A physiological method of protecting the joints is Active Alerted Posture in which the knee is held very slightly flexed. All the muscle groups are then in balanced contraction; that is to say, there is slight contraction of the quadriceps, hamstring, medial and lateral rotator muscles. In this position of alertness and readiness for movement, the strains of gravity are not thrown directly on the ligaments and joints themselves, but are absorbed in the already alerted muscles.

Complications which follow injury to the knee can obviously be avoided by appropriate immediate treatment.

Diagnosis

The exact diagnosis of an athletic injury to the knee can be extremely difficult and present many problems, even to surgeons with tremendous experience: but with an accurate history and careful examination it is usually possible.

Apart from the normal principles of examination applicable to any joint, the type of injury, any previous injury, the exact position of the tender points, the exact range of movement and the presence of a painful arc of movement, there are certain special tests. McMurray's rotation tests carried out in varying degrees of both flexion and extension are, if positive, pathognomic of a torn meniscus; but they are often negative when there is a complete bucket-handle tear of the meniscus. There is sometimes a peculiar sensation of springiness in the joint on full flexion. In such a case, the last two or three degrees of flexion are limited and the patient cannot take his full weight on the injured knee in the squatting position. If one forces flexion, one gets the impression that there is something between the joint surfaces limiting the movement. Similarly, in extension: the last few degrees may be limited by a displaced meniscus, thickening of the infrapatellar pad of fat and alar folds, or by a shortening of the medial collateral ligaments with associated spasm of the hamstrings, sartorius and gracilis. Compression and distraction test: this test is performed with the patient prone. The tibia is rotated on the fixed femur with the joint in varying degrees of flexion and extension. At the same time the tibia is compressed towards or distracted away from the femur. The object of the test is to prove whether an internal derangement is due to a tear of a meniscus or not, but we have not found it of much assistance. It has the advantage that it reminds the surgeon to examine the structures in the popliteal space.

The retracting cartilage, described by Watson Jones, is of great diagnostic significance. The medial meniscus tends to withdraw into the postero-medial part of the joint, leaving a furrow in the front of the meniscus where it lies between the front of the medial femoral and tibial condyles. When it is torn the meniscus may not retract and the furrow is filled with the anterior part of the torn cartilage.

For the differential diagnosis of meniscus injuries, Somerville[7] uses arthrography. He instils about 100 mls. of sterile air into the knee joint and takes three different exposures of each meniscus—the anterior horn, the middle portion, and the posterior horn. This is a most valuable aid in doubtful cases: if a meniscus is shown to be torn it can be removed while the patient is still under the anaesthetic.

In some cases it is sufficient to examine the joint under full general anaesthetic. Injury of the lateral ligaments, or cruciate ligament, can be diagnosed, especially if radiographs of the joint are taken when strain is thrown on the suspected ligament. The most difficult cases are those in which there may have been a slight contusion or tear of one of the meniscii, or an articular cartilage erosion. The position of these erosions varies with the type of in-

jury: in dislocation or subluxation of the patella, for example, the erosion is often on the lateral femoral condyle; an erosion accompanying a medial meniscus injury is on the trochlear surface of the medial femoral condyle.

Chondromalacia patellae often follows a knee injury particularly those involving the medial collateral ligament, as in ski-ing accidents. Localised tenderness on both sides of the patella with marked patello-femoral grating is pathognomic of this condition.

Direct injury

The effects of direct injury naturally vary according to the severity of the blow. Tissues may be mildly contused or so badly that they may die. If the blow falls on the articular cartilage of either the femoral, tibial, or patellar articular surfaces, the cartilage may become eroded.

If the injuries are trivial, with active treatment the knee should recover in a few days: but if there is extensive bruising of the tissues, recovery may take several weeks, even if the knee has been treated actively from the start.

The following case illustrates the effects of severe contusion and bruising from direct injury. The patient, who was riding in a steeplechase, was struck on the inner side of the right knee. He came in for examination three weeks after the injury. There was bruising on the inner side of the knee, with traumatic effusion in the knee joint itself, and extensive swelling, involving the inner side of the knee to the middle of the calf, from the seeping of haematoma. The knee was held slightly flexed and extension was limited by 20 degrees. Flexion was only possible through a range of 20 degrees and there was marked quadriceps wasting. The patient had been told that as a result of the blow, traumatic thrombo-phlebitis had developed. He had been advised that under no circumstances should active treatment be started because the thrombo-phlebitis made rest essential. The explanation of the injury was that the long saphenous vein had been ruptured and there was a large extravasation of blood which was causing a spreading oedema and tenderness of the tissues far remote from the original blow. With active treatment and very carefully graded exercises including graduated weight bearing, at first on crutches, the patient was completely symptom free in ten days. In fact he won the Grand National Steeplechase three weeks from the time he was first seen.

Blows on the patella often produce traumatic prepatellar bursitis (housemaid's knee). The force of the blow may cause contusion of the under articular surface of the patella and a subsequent chondromalacia patellae. The adjacent femoral articular cartilage and other soft tissue structures, such

as the infrapatellar pad of fat and the menisci, may also suffer direct injury. These conditions are often associated with synovitis, if the injury is mild, or even haemarthrosis if it is severe. If any part of the bone adjacent to the articular cartilage is so damaged that it is deprived of its blood supply it may eventually separate as a loose body.

Indirect injury

In indirect injury, whether it be a flexion, abduction, adduction, or hyper-extension strain, the synovia is often stimulated, with resulting effusion. If any of the structures inside the joint are torn or contused, blood vessels may rupture and cause haemarthrosis.

Flexion strain is liable to cause a tearing of the structures in the anterior part of the capsule, and compression and contusion of the intra-articular structures at the back of the joint. In a hyper-extension injury, on the other hand, the infrapatellar pad of fat and the alar folds are liable to be bruised. If the injury is severe, the tibial spine may be avulsed, together with the tibial attachment of the anterior cruciate ligament.

Rotation strains are liable to affect the menisci. Pure abduction strain usually involves the medial collateral ligament; adduction strain involves the lateral collateral ligament.

There is frequently a combination of two or three movements; for example, in a flexion strain there may be a degree of abduction as well as rotation. These injuries are considered under the 'Knee Joint Triad'.

Treatment of Traumatic Synovitis

Either a direct or indirect injury may cause synovitis which may be mild, moderate or severe. In mild synovitis the fluid is usually pure effusion; in moderate and severe synovitis the effusion may be bloodstained.

Mild degree of synovitis: usually there is a moderate amount of effusion and the athlete is able to take a certain amount of weight on the leg. Home treatment and physiotherapy should be started immediately and the patient advised to modify his activities. Organised physiotherapy should consist of some of the following:—massage, short wave, anodal galvanism, faradism, and gentle manipulation. Often mild synovitis recovers in two to three days and it is possible for an athlete to return to full activities within a week. Tender thickenings in the synovia must be dispersed and there must be full range of movement, so that the patient can do a full knees-bend without any pain, before he is allowed to go back to his sport.

Moderate and severe synovitis: the treatment of the two conditions is very similar, except that severe synovitis may demand a split plaster of Paris sup-

port, while moderate synovitis may need no more than gamgee and a crêpe bandage, or gamgee, felt and a crêpe bandage. Synovial effusions large enough to be obvious should be aspirated. This is done under strict aseptic precautions, usually by piercing the skin, capsule and synovia on the outerside of the suprapatellar pouch. To accomplish this without undue pain, 4 mls. of 2 per cent procaine or xylocaine are placed into the skin, subcutaneous tissues and the capsule, and if the effusion is bloodstained a large bore needle is employed.

In one case in which the synovitis was associated with the dislocation of the patella, 240 mls. of bloodstained effusion were removed at a session and exactly a week later a further 120 mls.

Following the aspiration it is usual to instil into the joint 1·5 mls. of hydrocortisone with 1000 units of hyaluronidase. In the early stages the patient does exercises only in bed. If walking is allowed the knee should be supported in a plaster cast and the weight of the limb carried entirely on crutches. Physiotherapy and home treatment should be started at once and carried out conscientiously two or three times each day. If the synovitis is so severe that a plaster of Paris support is indicated, it ought not to be split for twenty four to forty eight hours after aspiration; but home treatment and physiotherapy in the form of graduated, isometric exercises with faradism may be started at once. When split, the plaster is held by suitable straps and bandage so that it can easily be removed twice a day for home treatment and once daily for physiotherapy.

Treated along these lines, moderate synovitis may completely recover within a week. Thus with further graduated exercises the athlete is able to return to full activities within a fortnight. In severe synovitis it may be three weeks before there is complete freedom of movement without pain, and it may take a further three weeks before the athlete is able to carry out full activity. If recovery takes longer than six weeks it is probable that there is intra-articular damage such as a contusion bruise of the infrapatellar pad of fat or alar folds, or meniscus injury. There may even be damage to the articular cartilage and articular cartilage erosion. In severe haemarthroses repeated effusions may form in spite of several aspirations, because the synovial membrane is irritated by the blood which cannot be evacuated. A certain amount of blood seeps into the back of the joint and it is impossible to remove all of it at the first session. The residue causes a recurrent reaction; but gradually the synovial effusion becomes less and less bloodstained. Pieces of inspissated blood may remain, however; and if the clots are not broken up by the massaging finger, they irritate the synovia and the under surface of the patella, and produce recurrent synovitis or patello-femoral chondromalacia. This last complication

is very frequent after severe synovitis, especially where there is also medial collateral ligament injury.

We have stated that even severe synovitis without complications is usually completely symptom free within six weeks: but in order to achieve this result, the patient must have physical treatment every day and at the same time carry out home treatment conscientiously twice daily himself. It is only in this way that chronic recurrent synovitis is prevented.

Medial collateral ligament injuries

These may also be divided into mild, moderate and severe. Severe injuries are usually associated with complications such as damage to the meniscus or to the cruciate ligaments. They are therefore discussed in the section on 'the knee triad'.

Injuries to the medial collateral ligament often lead to so much chronic disability and a weak, loose joint, that the need for immediate treatment with suitable support cannot be overstressed. In mild strain, support in the form of a felt pad and crepe bandage after an injection into the tender point of the ligament of 4 mls. or 2 per cent procaine or xylocaine and hydrocortisone 1·5 ml. with 1000 units of hyaluronidase, may allow the patient to bear weight at once with the aid of a stick. Home treatment and physiotherapy are instituted immediately. By this method it may be possible to return the patient to full activity within a week.

Mild strain is usually not associated with synovitis. If there is synovitis, one should regard the injury as at least moderate, and it is important to X-ray the knee because a flake of bone from the upper femoral attachment of the ligament may have been pulled away. This avulsion is more common in severe injury. An X-ray taken with an abduction strain on the knee may show an opening on the medial side of the joint. It used to be common to find areas of calcification in this ligament after a tear, especially at the upper end (Pellegrini Stieda ossification): but now that injection therapy and active treatment are the fashion, the traumatic swelling is more quickly absorbed and this complication is not now so frequently seen.

It is of paramount importance to place the knee in a removable plaster of Paris cast in full extension, after an injection of procaine, hydrocortisone and hyaluronidase, as described above. Over the tender point a felt pad is placed before the plaster is applied. Exercises in the plaster should be started at once and they should be done at frequent intervals throughout the day. The plaster is removed once a day for physiotherapy.

When the damage seems to be severe, the patient should be anaesthetised

and radiographs taken with the knee forced into abduction. If the joint opens on the inner side more than 30 degrees, it is necessary to operate.

In all probability there are other injuries as well as that to the ligament, and these are described in the section on 'the knee triad'. If operation is not undertaken, the tender point in the ligament should be injected (as previously described) and the knee placed in plaster in full extension. With a combination of rest from strain, exercise without strain, good home treatment and physiotherapy, there is no reason why the moderate tear should not recover in a month and the severe tear, not requiring operation, in six weeks. The patient should be symptom free in half that period.

Lesions and injury of the menisci

Injuries, particularly tears, of the menisci occur as the result of flexion and rotation strain. Occasionally there may be a transverse tear of the medial meniscus at about the point where it is attached to the medial collateral ligament; and there may be an associated bucket-handle split of the lateral meniscus. There may sometimes be doubt as to which meniscus has been damaged, but if one pays attention to the mechanism of injury and the location of the pain and the tenderness, the diagnosis can usually be made. It may help to examine the knee under a general anaesthetic or to employ the X-ray arthogram techniques already described.

If a meniscus is torn it must be removed. No other treatment can guarantee a stable joint. At operation the surgeon should assess the stability of the ligaments and the appearance of the articular cartilage surfaces, including that of the under surface of the patella.

Undue laxity of the ligaments may be corrected by capsular pleating, overlay of the medial collateral ligament or by reinforcing this ligament with a semitendinosus tendon transplant placed over the ligament on the medial side of the joint. Repair of an injured cruciate ligament, discussed in the section on 'the knee triad', may also be undertaken.

Injuries to articular cartilage can be dealt with at the same time. If there is a large area of cartilage becoming separated, it may be necessary to drill the under surface and pin it into place, by an operation described by Smillie. Pridie, however, considers it sufficient to drill the underlying bone and replace the sequestrating piece of cartilage which will keep its place if the knee is held in firm extension after operation for three weeks. Provided there are no complications such as weak ligaments or osteochondritis dessicans, there is no reason why operations on the menisci should not be a great success so that the patient can use the knee for strenuous exercise two months after medial menisectomy and three months after lateral menisectomy.

Most orthopaedic surgeons now agree that it is futile to try to replace a torn meniscus by manipulation, even if it is carried out under full general anaesthetic. The result is usually to damage the meniscus by increasing the area of the split so that the torn pieces can rest between the surfaces of the condyles. If such a knee is later opened, one often finds not only this splitting of the meniscus but also considerable damage to the articular surfaces of the femur and tibia themselves.

It is reasonable, however, to make a gentle examination of the joint under an anaesthetic, provided the surgeon operates if he finds the meniscus is torn. If the meniscus is torn and there is some strong contra-indication to immediate operation, it is far better to leave the movements of the knee slightly limited. As the reaction in the joint settles down so the knee will gradually straighten: but if it is manipulated into full extension or flexion, further damage occurs as described. Although next day it may be thought by the inexperienced that the meniscus has been reduced by the manipulation, the muscle spasm will have returned and there will again be limitation of movement and obvious recurrence of the disability. The joint derangement, in fact, was never really corrected.

Sir Herbet Barker, the famous bonesetter, used to claim he reduced displaced menisci. His method, carried out under full general anaesthetic, was to flex the knee fully and at the same time to rotate it forcibly inwards. The result often was to complete the bucket handle tear of the meniscus and sometimes even free one end of the torn piece. With luck this would roll into the middle of the joint and not give trouble for many years. The majority of these cases, however, came up eventually for operation.

THE KNEE JOINT TRIAD

Surgeons often have to examine athletes who have sustained a combination of abduction, flexion and rotation injury. It is sometimes extremely difficult to determine whether or not there has been an injury to the medial collateral ligament, to the medial meniscus or to one of the cruicate ligaments. Certain clinical signs make involvement of one of the inter-articular structures a probability (vide supra).

There are two approaches to treatment, one conservative and the other radical. The conservative approach may be further divided into two philosophies. In the first, the joint is examined clinically and with X-rays (but not under an anaesthetic) in an attempt to assess the degree of ligamentous damage. If there is a large effusion it is aspirated under a local anaesthetic. After this the knee is placed in a long plaster and kept in it for a length of time

FIG. 2. Manipulation of knee. This is to demonstrate the importance of pulling the tibia well forward on the femur when manipulating the knee joint. This may be combined with rotary movement.

which varies according to the amount of damage to the ligaments. On the Continent this period is often as much as six weeks: and it is far too long. If the medial collateral ligament is thought to be partly torn, the plaster ought to be split at the end of a week. From the first day the patient should have physiotherapy and home treatment several times each day. After the plaster is split, he should be allowed to flex and extend his knee with the heel of the leg on the bed. In this way no rotary movements take place. After a week he should be allowed to walk in the plaster cast with the aid of crutches, taking a moderate amount of weight. Under this regime, which allows gradual flexion of the knee with the split plaster removed, most patients recover full movements in two to four weeks: but it may be a further two to four weeks before strenuous exercise can be taken.

The second philosophy is really midway between the conservative and the radical approach. In doubtful cases the knee is examined under a general anaesthetic. Before the anaesthetic is given tender points are noted. When the patient has been anaesthetised, the surgeon assesses the mobility of the joint and X-rays are taken with strain applied in various directions, in order to determine the integrity of the ligaments. McMurray's rotation tests are also

FIG. 3. Manipulation of knee joint. Freeing the patella from side to side.

performed to see whether there is injury of a meniscus. If there is an obvious rupture of the medial collateral ligament or an associated meniscus tear, the surgeon operates at once: if not, he applies a plaster cast which the patient keeps on for varying lengths of time. If he is in doubt and there is no effusion in the joint, he can perform an arthogram by the Somerville technique, and then proceed with operation or simple immobilisation, according to the result.

The radical approach is that of O'Donoghue[8] of Oklahoma City who considers most moderate and severe injuries to the knee joint usually require immediate operation, especially where there has been a combined abduction or adduction strain with flexion or rotation. He explores the joint, removing damaged menisci, and reconstructs the cruciate ligaments if they have been torn.

O'Donoghue[12] has performed many hundreds of operations on severely damaged knees and considers that in those cases in which there is:—

(1) obvious ligamentous injury giving rise to joint instability
(2) haemarthrosis
(3) probable tear of one of the menisci

FIG. 4. Manipulation of knee. The patient is lying prone. In this position it will be found the manipulator has great control over the knee movements in this position.

operation should be carried out forthwith. After preliminary confirmation of the findings under full general anaesthetic the joint is explored, damaged menisci removed and torn cruciate ligaments reconstructed. These ligaments are repaired by end to end suture if this is possible. A torn anterior cruciate ligament may be replaced by a strip of fascia lata taken from the lateral aspect

of the thigh and threaded through drill holes. In posterior cruciate rupture the semitendinosus tendon is employed in the reconstruction.

In moderate to severe cases, operative reconstruction of the knee gives quicker and more definite results. It is only by operation that the severely damaged knee has a chance of being restored to normal.

Acute contusion of the infrapatellar pads of fat and alar folds

In severe injuries, especially direct ones and those in which there has been an acute hyperextension wrench, these structures may suffer severe contusion which results in prolonged disability. The knee may be so painful that the surgeon feels bound to explore it.

Occasionally a piece of fat becomes crushed, a condition called 'acute fat necrosis'. This is associated with swelling, acute pain localised to the area, increased heat of the overlying structures and varying degrees of limitation of movement. Immediate operative removal is the best treatment.

Osteochondritis and chondromalacia patellae

These two conditions may be considered together, because an erosion on the trochlear surface of the medial femoral condyle is often accompanied by a large erosion on the medial aspect of the articular surface of the patella. These lesions often complicate a medial meniscus injury due to flexion and medial rotation of the femur on the tibia. On the other hand, where there is a tendency for the patella to dislocate laterally, the areas of erosion affect the lateral condyle of the femur and the lateral articular surface of the patella. The treatment is to smooth off the roughened articular surfaces. In certain cases it may be possible to replace a large piece of articular cartilage by Smillie's or Pridie's method.

In the older athlete, over thirty, it may be wiser to carry out a patellectomy if there are signs of early osteoarthritis of the joint. Many athletes have been able to carry on in first class cricket and even in football without a patella.

Injuries to the lateral collateral ligament

These do not differ from other similar ligamentous injuries, except that the ligament is very close to the biceps tendon. It is not attached to the lateral meniscus so that when it is injured there is not necessarily damage to the meniscus. Medial collateral injuries of any severity, on the other hand, nearly always involve the medial meniscus.

Bursae

In injuries around the knee joint it must not be forgotten that certain bursae

in relation to ligaments and tendons are liable to be involved in an inflammatory state. Occasionally a constitutional condition becomes superimposed on the traumatic state and makes recovery slow. Occasionally operative removal of the inflamed bursae becomes necessary.

Popliteus tendon

Injury to this tendon often accounts for weakness of medial rotation of the tibia on the femur and pain on forcing medial rotation. If the tendon is ruptured when the patient is standing with the foot pressing on the ground, the tibia can be displaced backwards on external rotation. Operative repair is possible if performed immediately after injury but is difficult at a later date. The chronic sprained knee is considered under complications.

STRAINS AND TEARS OF THE CALF MUSCLES

Partial tears of the calf muscles may affect some parts of the gastrocnemius, the soleus or the plantaris. It used to be considered that the plantaris muscle was the muscle most involved: but in ruptures of the tendo achilles the plantaris tendon always remains intact.

The history of injury is usually characteristic. The patient, often over thirty-five years of age, performs a sudden quick step as when making a shot at tennis. He feels as if he had received a hard blow with a cane or tennis ball on the back of the upper calf muscles. The position of tenderness varies, and by noting it, and the location of the pain on passive stretching, one is able to recognise which muscle is damaged. Unless the concomitant traumatic effusion is quickly dispersed by home treatment, physiotherapy and local procaine, hydrocortisone and hyaluronidase injections, it tends to organise into tendon thickening at a slightly lower level than the original injury or sometimes close to the musculo-tendinous junction. Once these thickenings have formed, it may take many weeks of intensive treatment to disperse them. If the haematoma is large and localised one may have to aspirate it or express it, especially in the early stages. This may save weeks of treatment.

By the active approach the patient should be symptom free in two weeks and able to carry out full activity in four weeks. He should learn Active Alerted Posture, as it is often the sudden strain placed on an atonic unalerted muscle, as the patient stands flat footed, which causes the original tear. Local bandage support is necessary and a sponge rubber heel is of considerable help during the period of rehabilitation.

Rupture of the tendo Achilles

If the tendon ruptures completely, the break is usually two inches above the insertion. In many cases it may be difficult to be sure of the diagnosis because the tendon has ruptured inside the fascial covering which, if it is intact, disguises the discontinuity. This injury occurs as the result of a sudden

FIG. 5. Manipulation of ankle and foot. The patient is lying prone. In this position it will be found that the manipulator has complete control of the movements at the ankle and tarsal joints.

movement, in sports like ski-ing, tennis, or hurdling, or in more everyday incidents like stepping up or downstairs suddenly, or when a foot falls through a wicker chair. A good diagnostic test is to ask the patient to stand on the affected foot with the contralateral foot off the ground. He should then try to rise up on the toes of the injured side, supporting all his body-weight on the ball of the foot and with the heel off the ground. This is impossible in any complete rupture of the tendo-Achilles but possible in ruptures of the plantaris or soleus alone. Another test is Campbell Thompson's. The patient lies on the couch prone with the heel over the edge of the couch. If the calf muscles are normal, on pinching them the heel rises, but if the tendo-Achilles is ruptured there is no movement of the heel upwards at all.

Treatment must be by operative repair. This is the only way to be certain of a satisfactory result. The fascial covering which has no synovial lining and therefore is not a true tendon sheath, is usually found to be intact

FIG. 6. Manipulation of the ankle joint. This demonstrates the importance of pushing the tibia backwards on the talus when manipulating the ankle joint for adduction sprains. In these sprains the body weight passes forward so that the tibia rides forward on the talus, a slight subluxation occurring. This manoeuvre replaces it with a thud.

and the ends of the ruptured tendon are only apparent when it has been opened.

The frayed ruptured ends should be opposed as accurately as possible and fixed with chromic catgut.

After operation the foot, ankle, and leg are supported in a plaster cast with the heel elevated, but the wound must be dressed on the fifth day, so that any haematoma can be detected and removed. Active isometric exercises should start in the plaster immediately but the patient must not put weight on the leg for six weeks. He is gradually returned to activity with the help of physiotherapeutic measures but it may be six months before he is ready to resume vigorous competitive sport.

INJURIES TO THE ANKLE JOINT

Sprains of the ankle are usually due to an indirect force which causes an adduction and inversion strain of the foot on the ankle, as when one 'ricks over' the ankle on some uneven surface. The talus is tilted outwards and the foot inwards. If the patient is standing or walking slowly, probably only the anterior fibres of the lateral collateral ligament are strained. However, if he is running when the foot adducts and inverts there is a second stage of the injury as severe strains are thrown on the ankle joint and talus by the motion of the body forwards. The talus is broader in front than behind, so that the tibia skates forwards on the tilted talus and bruises the outer edge of the articular cartilage. It may even plough up the surface of the articular cartilage and a piece of the underlying bone, an accident which may lead to osteochondritis dessicans. In certain cases of moderate and severe sprain, the tibia is fixed against the broader anterior edge of the talus. Before full painless movement can be established this subluxation must be reduced. The reduction is performed by traction on the heel with counter-traction on the lower end of the tibia, the anterior surface of the lower end of the tibia being simultaneously pushed backwards. As the bones go back into place there is often a 'clonk' which is both heard and felt by the patient and the surgeon.

When the foot twists over while the patient is in motion, the head of the talus is forced inwards and downwards on to the spring ligament and tibialis posticus, which, with the deltoid ligament, sometimes suffer a direct contusion injury on their deep aspect, being jammed by the head and neck of the inner border of the talus. The surgeon should bear these possibilities in mind; for if these conditions are not treated, the patient later gets symptoms of strain of the longitudinal arch, with tenderness and local thickenings. All this can be avoided if it is realised that the structures on the inner side must

be included in the programme of treatment. If there is deep tenderness when the foot is first examined, injection of procaine, hydrocortisone and hyaluronidase must be given as soon as possible.

Sprains of the ankle may be classified as mild, moderate or severe. Ellis,[22] who is in charge of the out-patient accident department at the Leeds Infirmary, and who treats some eight hundred cases of sprained ankle each year stresses the importance of localising tender points and describes an injury to the inferior extensor retinaculum. This structure may be involved if the tender point is situated half way between the tip of the lateral malleolus and the styloid process of the fifth metatarsal bone. The peroneus brevis tendon is attached to this latter bony point and on cine-radiography, in the movement of adduction and inversion of the ankle, it can be seen that this point is situated at the circumference of an arc of a circle in which the foot, with the talus, rotates round the ankle joint. In some cases of injury there may be an associated fracture of the styloid process of the fifth metatarsal bone because so much force is applied to this bony point (Robert Jones).

In mild sprain it is usually only the anterior fibres of the lateral collateral ligament which are stretched. There is no joint effusion and treatment can

FIG. 7. Testing for sprain of the posterior fibres of the lateral ligament (talofibular ligament). Everting the forefoot while adducting hind foot thus stretching the posterior fibres of the lateral ligament. Strain of these fibres gives pain up the posterior margin of the fibula on kicking a ball on the inner part of the foot.

be expected to get the ankle back to normal in a matter of two to three days.

In moderate sprain there is not only a rupture of some fibres of the anterior and medial fasciculus of the lateral ligament but also an effusion into the joint.

Severe sprain may be associated with complete rupture of the lateral ligament as demonstrated by tilting of the talus on forcible inversion. The posterior fasciculus (posterior talofibular ligament) of the ligament may be also involved, as shown by pain when the forefoot is everted on the back foot. If this fasciculus shortens as it heals, the patient is unable to kick a football without considerable pain. This is because in the action of kicking the forefoot is forced outwards on the back foot and the ligament is stretched. It is important to appreciate this point, especially if the ankle is being manipulated for stiffness associated with a chronic sprained ankle. In such cases there

FIG. 8. Manipulation of ankle joint. This demonstrates the importance of forcibly adducting the ankle at the subtaloid joint when manipulating for chronic sprained ankles. This movement frees the joint and stretches the middle and post posterior bands of the lateral ligament.

may be varying degrees of capsular rupture and synovial tearing. The greater the damage to the synovia, the more effusion there will be.

The decision whether or not to operate depends on the patient and his requirements. If there is obvious ligamentous rupture in a young, active patient such as a professional footballer, it is best to open the joint under strict asepsis, using a tourniquet. It is possible then to examine the interior of the joint, specially the articular surface of the talus. Blood, clot and traumatic effusion can be evacuated and the ruptured ligamentous fibres orientated and held in position by catgut suture.

Patients who have moderate or severe sprains with synovial effusion but no rupture of the ligament, are best treated by aspiration, support, graduated exercises and physiotherapy with home treatment. They are best placed in a comfortable removable walking plaster. Those who have had an operation should perform strong isometric movements in the plaster. The muscles can be stimulated by faradism given above the plaster or through windows. The plaster is removed after a fortnight so that the stitches can be removed and the wound dressed. Provided there is no articular cartilage damage the patient can start non-weight-bearing exercises out of plaster at once. He is allowed to

FIG. 9. Manipulation of the ankle. The tibio-talo-calcaneal articulations are distracted. With the patient's knee in the operator's axilla a distraction force is exerted by flexing the patient's knee against the operator's elbow. The operator's other hand is free to produce tilting movements of the joints.

soak his ankle in a hot bath and to start walking in the plaster, taking most of his weight on crutches. He maintains the removable plaster until there is full range of active movements without weight bearing. This usually takes about four weeks; and if he has been carrying out strong isometric contractions and having faradism daily he will probably be able to walk with a felt and bandage support and a stick. In a further fortnight, making six weeks in all, he is usually sufficiently recovered to start training again. Thus, provided there is no articular damage he will be fully recovered in eight to ten weeks.

The care of the chronic sprained ankle should follow the general line of treatment for stiff joints and loose joints as described in Part III, Section I, Chapter 9.

If pain and stiffness persist two months after injury, adhesions probably have formed and the foot and ankle require manipulation under full anaesthesia. Care is taken to be certain the tibia is pushed well back on the talus and the forefoot made freely mobile on the back foot. The posterior talofibular ligament is freely stretched by abducting the forefoot at the same time as adducting the hind foot.

FIG. 10. Manipulation of the naviculo-cuneiform-metatarsal joint. The manipulator's index finger acting as a fixed fulcrum while the fore part of the foot is forced into plantar flexion by the other hand. The movement should be made as a quick flick of low amplitude, rather than a wrench. There should be no 'follow through' of the movement.

The treatment of sprain of the mid-tarsal, tarso-metatarsal, metatarso-phalangeal and interphalangeal joints should follow the general rule. Care is taken that the longitudinal arch with the transverse metatarsal arch are suitably supported until the muscles have recovered their full tone, power, extensibility and flexibility.

Plantar fasciitis

This condition is common in athletes, especially those with high longitudinal arches and may be due to postural and occupational strain of the leg and foot muscles alone. A rheumatic or gouty diathesis may also be present and often there is a history of a direct blow on the heel or foot. If the condition becomes chronic and is of long standing, a calcanean spur may form in relation to the origin of the short plantar muscles from the tuberosity on the under surface of the calcaneus. Treatment should include elimination of any septic or metabolic diathesis if present, sponge rubber heel pads, hydrocortisone injections, home treatment and physiotherapy. Most cases clear up in a few weeks without surgery.

Acute foot strain

This condition is frequently found in athletes with high arched feet or those with flat rigid feet. Often the condition occurs suddenly after a bout of strenuous exercise in the middle of the season. As the result of strain, the spring ligament, the tendons of the tibialis posticus, flexor hallucis longus and the peronei are particularly liable to be involved. Stenosing tenovaginitis is common and if not cured by rest, hydrocortisone injections, home treatment and physiotherapy, may necessitate operation. In certain cases adhesions form and manipulation of the ankle and foot under general anaesthesia followed by graduated exercises, physiotherapy and home treatment produce a cure. Some form of foot support is usually necessary.

REFERENCES

1. Gray's Anatomy (1960).
2. Galeazzi, R. (1927). 'Clinical and experimental study of lesions of the semilunar cartilages of the knee joint.' *J. Bone Jt. Surg.* 9, 515.
3. Helfet, A. J. (1959). 'Mechanism of the medial semilunar cartilage and their management.' *J. Bone Jt. Surg.*, May, Vol. 418, No. 2, p. 319.
4. Helfet, A. J. *Ibid.*
5. Last, R. J., Personal communication.

6. Kaplan, E. B. (1961). 'The fabellofibular and short lateral ligaments of the knee joint.' *J. Bone Jt. Surg.*, March, Vol. 43A. No. 2, p. 169.
7. Somerville, E. W. (1946). 'Air arthography as an aid to diagnosis of lesions of the menisci of the knee joint.' *J. Bone Jt. Surg.*, July, Vol. 28A, No. 3, p. 451.
8. O'Donoghue, D. H. (1955). 'An analysis of end results of surgical treatment of major injuries to the ligaments of the knee.' *J. Bone Jt. Surg.*, Vol. 37A, No. 1, January, p. 1.
9. Ellis, M. (1955). 'Soft tissue injuries of ankle and shoulder'. *Physiotherapy Journal*, August.

8

A CRITICAL SURVEY OF METHODS EMPLOYED IN THE TREATMENT OF ATHLETIC INJURIES: THE DIFFICULT CASE TO CURE

THERE are several ways of treating an injury and obtaining the same result. Obviously the object should be to extract the maximum amount of benefit from each.

It has been stressed more than once that, as the result of injury, infection, reaction to toxins, metabolic disturbance, or reaction of degeneration, the tissue response is ultimately one of fibrosis. The structures involved never return completely to normal. The tissues pass through two physiological phases, the negative and the positive; and through three clinical stages—acute, sub-acute and chronic. If the patient is given proper treatment, the chronic stage can usually be avoided; but it may be extremely difficult to be certain in which phase or stage the condition is, at the time of the examination. In treatment, beneficial effects may only show when the final fibrotic process has been established.

A particular treatment may apparently be ineffective, and in fact the condition may appear to be worsening if the injury is in the negative phase. Both doctor and patient may lose faith when, in reality, the treatment is helping to hasten recovery. For example, in sub-acute sprain of the metacarpophalangeal joint of the thumb of a boxer, with tender thickened synovia and capsule, active treatment at first makes the joint more swollen and more painful; but gradually, if doctor and patient persevere, a complete cure is established. During the exacerbation, the thumb has to be rested from all forms of strain and protected by suitable support from all jars and knocks; the morale of the patient has also to be supported by constant encouragement.

In the acute stage, rest from strain is the essential principle. If the part is painful, warmth and analgesics are also essential. Often at this stage short-wave diathermy, anodal galvanism, or interferential therapy are all that the patient can stand. It may be possible to add gentle effieurage and to persuade him to move the injured part very gently, after sponging it with hot water, through as full a range of movement as he can manage. Hydrocortisone injections help to prevent excessive fibrosis and thus facilitate recovery.

During the sub-acute stage more vigorous physiotherapy, including very gentle manipulation, may be started. This stage can be recognised by the loss of pain at night and slight, but definite increase in the range of movements.

If the patient is first seen in the chronic stage, vigorous methods may be started immediately, but one must be careful to avoid an acute aggravation of symptoms. Some reaction to physical treatment is to be expected, but it should not go beyond pain lasting a few hours. If the patient is kept awake and there is increased swelling and muscle spasm either the physical treatment has been too vigorous or there is some complicating factor, such as a toxic focus, present.

On the whole it is wiser to start with gentle daily treatment, in order to improve the tone of the muscles and to clear away thickenings and tissue oedema before one attempts to break down adhesions. Manipulation under an anaesthetic in the presence of wasted muscles and tissue oedema may successfully break down adhesions, but, as a result of the marked reaction, the adhesions will tend to re-form.

If even gentle physiotherapy causes a severe reaction it is not necessarily evidence that the diagnosis is wrong. The cause may be a toxic focus or some metabolic disorder like gout.

There are in modern life many stresses upon athletes apart from those of their sport. A woman athlete who is a busy housewife with a husband and children may begin quite suddenly to show the effects of the combined strain of competition and domestic cares. All she needs as a rule, is rest, and to make sure she gets it, she should be sent to bed for a short time. If physical treatment is available, so much the better.

Similarly, any athlete who repeatedly breaks down from a muscle or joint injury often recovers quickly if he changes to another sport, which make different demands on his muscles.

Weight-training

There is no question that graduated weight-training exercises are valuable and even essential in suitable cases. The danger, however, is that the weight or the resistance used may be too great so that the patient's muscles and joints are put under strain. This applies particularly to injured knee joints in which the ligaments are stretched and the muscles wasted. If the patient is made to lift weights strapped to his foot before the muscle tone returns to normal he is likely to delay rather than promote recovery.

The weight or resistance placed on muscles should always be just less than the maximum which produces strain. Excessive exercise always produces further wasting of muscles, swelling of joints, increased pain, restriction

of movements and early morning stiffness: therefore the patient must be closely supervised and his exercises carefully graduated to his individual needs.

Seyle[1] has brought forward his theory of 'adaptation' and stress syndrome. If the cumulative effect of the ordinary wear and tear of life is the cause of a breakdown, the patient's everyday life must be altered for treatment to be fully beneficial. This explains why, if the patient is ambulatory and carrying on with his ordinary daily life, treatment consisting of physiotherapy with manipulations and exercise will often have no positive effect. In the case of severe injury if the patient is obliged however to continue with an active professional life, this plus any form of physical treatment may be detrimental and can produce a reaction with aggravation rather than alleviation. In a case of this type, if the everyday stress of a busy life is substituted by rest in bed, a good effect may be seen immediately. It is while resting that physical treatment, including gentle manipulations and very gentle remedial exercises carried out at intervals during the day will have greatest benefit. It is sometimes extremely difficult to convince the athlete that he or she must relinquish for a while all the ordinary activities of daily life. If rest from strain is to be achieved it must be made clear to the athlete that recovery will be quicker if he gives himself up to the combined beneficial effects of rest and physical treatment including manipulations and gentle remedial exercises. With firmness of conviction he can usually be persuaded to adopt this regime from the start.

The advisability of employing physical methods including manipulations and exercises, will be questioned by some but a time comes, nevertheless, when a patient must start to move and prepare himself to return to his everyday life. It is then that 'purposeful aggravation' in the form of modified and gentle physiotherapy, including exercises is helpful to determine whether the case is in the negative or positive phase.

Whenever rest in bed is indicated the patient should be allowed to move about as freely as possible and to carry out special exercises daily in order to prevent stasis of fluid, venous thrombosis, wasting of muscles and stiffness of joints. By supporting the extremities, rest from weight bearing allows exercises to be carried out without strain.

In recovering from any condition whether injury or disease, active movements should be carefully graduated.

The sequence of graded active exercises is:—

A. Without weight bearing or strain:

1. With the aid of gravity.

2. With a sling.
3. Exercises in water.

B. With strain:

1. Against gravity.
2. With graduated weights.
3. Against strong resistance.

C. Weight bearing exercises:

1. Gentle.
2. Moderate.
3. Strenuous.

When a patient is first allowed out of bed after an injury it may be difficult for him even to take a step. On the other hand if he has been carrying out bed exercises including pushing down against the foot of the bed in order to keep his leg muscles fit, he should be able to return to his normal activity within a few days.

The distance the patient can walk each day without tiring is a good guide to the speed of his recovery. Excessive exercise, like an overdose of medicine, may cause a reaction. During convalescence the athlete must try to keep just under his maximum to prevent a setback.

If muscles of the part are atonic and easily tired, as may occur in a person getting out of bed after an illness, the venous pump is faulty and graduation of weight bearing exercise is essential.

For maximum benefit from physical methods, treatments should be given individually. By noting the significance of the physical signs and symptoms it is possible to tell in most cases exactly through which phase or stage the condition is passing, and therefore to advise the type of treatment most beneficial. The reaction of the muscles to faradic stimultion can sometimes be a good guide to the phase through which the condition is passing. If there is some contributory constitutional factor inhibiting recovery, the muscles tend to stutter, fasciculate, and contract badly.

Physiotherapy can be overdone, but the active athletic patient is usually able to benefit from it as often as twice a day, while at the same time carrying out a course of home treatment and remedial exercises.

Evaluation of various forms of treatment:—

1. *Rest*

It is obvious that rest for too long a period is a waste of time. Rest from

strain can usually be advantageously combined with physiotherapeutic measures, including gentle remedial exercises. However, where there is an acute condition or excessive reaction to treatment, a short period of rest in bed is indicated without more active treatment. This should not exclude free supported movements of the mobile parts in bed once or twice daily.

2. *Baths*

It is seldom that contrast baths or hot baths cause a reaction. In conditions where there is increased tissue tension hot baths will increase the blood supply and by further increasing the tension make the affected part more painful. In cases of this type patients often state that hot baths or Turkish baths have made them feel worse. Some of these may have a toxic focus such as a gouty diathesis or gross degenerative tissue changes. The increased blood supply from the hot baths then tend to aggravate. If this type of patient would stay in the bath for a shorter period and combine the baths with physiotherapy, diet and medicinal treatment, the effect usually would be beneficial. The patient is then able to stay in the bath for longer periods as improvement becomes apparent.

3. *Country rehabilitation centres*

Treatment at a centre of this type can be ideal for the athlete with the chronic complications of injury. The severely injured athlete often derives great benefit from organised treatment obtained at such a centre. There the patient rests from his ordinary everyday life, and at the same time is able to have various forms of physical treatment often including manipulations. If overweight, he is placed on a diet and by losing weight will reduce strain on his weight bearing joints.

At some centres patients perform exercises under water and these are particularly good for countering the effects of injury. The exercises are then performed without strain as the buoyancy of the water reduces the body weight to about one-tenth. Special exercises can be given which move the pelvis and thigh on the trunk and strengthen the prime fixing muscles of the trunk, pelvis and thighs.

However, the determined patient, if not seriously ill, is usually able to obtain benefit by disciplining himself at home. He must cut down his activities but by combining a dietary routine with modified rest, plenty of sleep and physiotherapy, including manipulations, he is able to remain ambulatory and carry on with most of his ordinary daily routine.

4. *Massage*

At the present time strong conflicting views are held by different authorities as to the real value of massage. Justifiably some patients will state that only deep massage of the Swedish type suits them, whereas others will blame deep massage for the aggravation of their condition.

It is obvious that there must be different types of massage for different conditions. Also the same condition may require a different type of massage at varying phases or stages in its progress. For example, in a recent traumatic capsulitis of the shoulder joint effleurage and gentle kneading only may be indicated and tolerated, whereas in the established chronic traumatic capsulitis only deep friction will be of benefit. Massage given gently can relieve tissue tension but deep massage causes an escape of fluid into the tissue and this helps in softening tender thickenings and adhesions in the chronic stage.

It would appear that there is a tendency at the present day by many physical medicine experts to forget the benefits derived from the various types of massage in favour of short wave diathermy and other forms of physiotherapy.

Most cases of injury will benefit to a greater extent if physical treatment terminates with a short period of appropriate massage and gentle manipulations.

At a meeting of the British Orthopaedic Association[2] in 1955 a motion was set before the meeting stating that 'Lucas Championère was right'.

Lucas Championère[3] was at his prime in Paris about 1850 and he stated that in all fractures, treatment of the soft tissues was of the utmost importance and should take preference over everything else. Of course, by modern standards, this is a fallacy especially in fractures of long bones of the weight bearing lower extremity. However, by methods advocated in this book immobilisation of the fracture for the desired time should not interfere with the treatment of the soft tissues to a greater extent than is essential for adequate consolidation of the fracture. Isometric exercises, faradism and massage can often be combined to run concurrently with the necessary period of immobilisation.

5. *Manipulations*

To many, manipulative manoeuvres imply the use of force under an anaesthetic, but the osteopathic physician employs gentle manipulative methods to most of his patients with benefit.

At a combined meeting of the Orthopaedic and Physical Medicine sections of the Royal Society of Medicine on the Role of Manipulative treatment one of the speakers thought that there must be a common ground of agreement between the osteopathic physician, who manipulates frequently and the

orthopaedic surgeon who performs manipulations infrequently and then only under an anaesthetic.

First of all let it be stated and understood that manipulative treatment must be properly performed to come to any conclusion. The following criteria must be adhered to for this form of therapy to do good.

(a) The manoeuvres must follow those taught by Mennell.
(b) There must be no septic or toxic state present.
(c) Reactions must be controlled and minimal.
(d) Whether manipulations are carried out with or without a general anaesthetic the principles should be the same.
(e) Even if manipulations are to be performed under a full general anaesthetic gentleness of handling must be stressed. There is an optimum period of time in the course of recovery that it should be carried out.

Most cases of recent injury, without complications to muscles and joints, are improved by gentle daily manipulation. It must be realised also that a contusion strain of, for example, the back of the hand can, by throwing the muscle out of balance, initiate symptoms and signs of strain in the muscles of the elbow, shoulder, shoulder girdle and cervical spine. In the early stages the patient is often not conscious of this but in some cases the early signs precede the symptoms and the observant surgeon will note them. Gentle manipulation to ensure full movements of the elbow, shoulder and cervical spine, is of paramount importance if complications such as the symptoms of a tennis elbow are to be avoided. It may even be that in the middle aged athlete there is present an unsuspected cervical disc degeneration which is gradually or acutely aggravated by strain thrown on it by an injury to any part of the upper extremity.

Manipulation under anaesthetic should play an important role in many cases of chronic injury and no patient should be labelled as a hysteric especially in relation to a knee injury without first subjecting the joint to an examination and controlled manipulation under anaesthesia.

Gentle manipulations should play an important part in the treatment of osteoarthritis with signs and symptoms of the early stages. Manipulations under general anaesthesia may be important in establishing as satisfactory a result as possible after a period of home treatment, physiotherapy and injections. The breaking down of terminal adhesions ensures often a more lasting benefit; if this is not carried out the symptoms tend to recur after a short time.

6. *Faradism*

There are some patients who cannot stand electrical stimulation of the

muscles and this idiosyncracy must be respected. These patients are often young children, fat women and gouty men. As already stated faradism is seldom given properly and the great benefits accruing from it, especially in the early stages following injury, are not realised or understood. It should never take the place of graduated exercises but should run in conjunction with them. There are many former athletes who have developed osteoarthritis of their hip and knee joints in middle age and find that a self treatment of fifteen minutes daily helps to keep the condition in check. There are many influential medical men who still consider faradic stimulation a waste of time and unnecessary. Of course, as has been said it should never take the place of active exercises but it is found by many patients to remove the stiffness and tired feeling produced by exercises. This is understandable as by artificial stimulation of the muscles without body weight no strain is put on the muscles, relative increased blood supply to the part is produced and waste products removed.

7. *Short Wave*

This supplies deep heat, but it would seem that the many physiotherapists are depending on results from this form of treatment rather than the use of their own hands by massage and manipulation.

8. *Ultrasonic*

In some cases in which pain is a predominant factor it would seem to reduce it but in the experience of most experts the fantastic claims made for it are not substantiated.

9. *Operative Procedures*

Some conditions do not recover quickly in spite of good conservative active treatment and full co-operation on the part of the patient. In certain specific conditions operative methods often give a better result and save an indefinite period of convalescence.

Indications for operations are becoming more defined, but if there is any question of doubt a compromise can be made in giving the patient a definite period of conservative treatment to test its effect. The conservative regime must however be intensive. The right amount of rest and exercise is adhered to strictly and the patient carries out his home treatment in the minutest detail as does also the physiotherapist and other members of the team. If after this definite period there appears to be no improvement or doubt as to the ultimate result further delay is procrastinating. In the healthy athlete the operative risk is obviously less than in the elderly. In certain cases by a stroke

of the knife the negative phase is eradicated so that the positive phase of healing is able to commence at once. Operative measures are definitely indicated in the following particular conditions:—

(a) In all compound or open wounds especially associated with fractures.

(b) Certain unstable fractures of long bones. This also applies to delayed or non-union in certain fractures, for example carpal scaphoid and medial malleolus.

(c) Injuries showing the presence of large extravasation of blood with tissue damage.

(d) Unstable joints due to injury in which there has been a rupture of a ligament or injury to part of the articular cartilage, meniscus or disc. This applies to certain definite conditions of the vertebral column.

(e) Recurrent dislocations of joints especially the shoulder.

(f) Certain cases of ruptures to muscles and tendons particularly in weight bearing ones such as the quadriceps expansion and tendo Achilles.

Difficult cases to make symptom free quickly

There are a group of cases in which of necessity there must be a lapse of time before they become symptom free. In many of these, operative procedures could be an alternative approach but even if resorted to, the patient may have to undergo a long period of rehabilitation.

Cases coming under this category are:—

(1) Contusion and bruising of bone and cartilage. Unless an active and vigorous approach is applied to these injuries at the commencement tender painful thickenings form especially in the hand and foot where there are tough fascial coverings. Often a joint will be said to be chronic and painful because of some loose intra-articular cartilage. This may be seen in a painful acromioclavicular joint in which a piece of the articular cartilage is shed and is lying loose in the joint. This, of course, may be one of the factors in producing a painful joint but it is my belief that many of these are due to the late effects of contusion of the adjacent articular cartilages, and that if the joint were treated vigorously from the beginning with the purpose of removing the effects of bruising, they would become free from pain without operation.

(2) Tears of certain muscles at their origin such as the adductor longus near the symphysis pubis; the tendo Achilles at the musculo-tendinous junction.

(3) Certain cases of stenosing tenovaginitis. Stenosing tenovaginitis of the long head of biceps brachii can account for the prolonged period of disability in many cases of capsulitis of the shoulder joint.

(4) Certain joint conditions where there has been a degree of stretching of the ligaments without actual rupture.

Although the condition of an injury as it presents itself is studied carefully, and appropriate treatment is given accordingly, it is imperative at the same time to consider all the possible complications that could arise in any particular type of injury and to take the necessary precautions to avoid these complications. For instance in the case of wrist injury it is possible that there may be involvement of the elbow and shoulder joints especially after a fall on the wrist. Naturally these joints must be examined and the appropriate treatment given if they are found to be involved.

Apart from stressing the importance of patients being conscientious in carrying out their home work, physiotherapy must be given throroughly so that a full treatment lasts up to an hour. In most cases it includes gentle manipulation with the purpose of increasing the range of involuntary movements as well as the voluntary. Sometimes despite vigorous treatment, adhesions form and at a later date, usually about two months after the injury, a controlled manipulation under pentothal becomes necessary to break down these adhesions.

Many cases need time to recover as well as several types of treatment. Treatment must be given conscientiously, daily and combined with the enthusiastic carrying out of home work. One realises only too often that for certain chronic conditions as many as fifty to one hundred treatments may be needed. The patient cannot expect to recover with only a few treatments. However, in these cases by the careful study of tissue reactions and the experience gained through perseverance with such cases, the patient can be assured that his condition will ultimately recover completely.

REFERENCES

1. Selye, H. (1950). *The Physiology and Pathology of Exposure to Stress*, 1st ed. Montreal: Acta Inc.
2. Perkins, G. (1955). 'That Lucas-Championère was right'. Debate at Brit. Orth. Assn. meeting in Manchester. *J. Bone Jt. Surg.*, 37B., p. 719.
3. Perkins, G. (1955). *Ibid.*

9

COMPLICATIONS FOLLOWING ATHLETIC INJURIES

We have stressed, it is often possible to avoid certain complications following injury. The doctor, if he anticipates them, can forestall them by appropriate precautions and active treatment.

Some authorities recommend that the only physical treatment for the first forty-eight hours should be postural drainage and firm pressure protection and support by wool, bandages or plaster. We believe this to be a loss of valuable time, because the traumatic effusion is more easily and quickly absorbed early on, when it is in the fluid stage, than later. When the symptoms are chiefly due to traumatic effusion in the subcutaneous tissues, without extensive tearing, the patient will recover almost completely within forty-eight hours by active home treatment alone.

The immediate complications may be classified under the following headings:—

Venous Thrombosis

The femoral and saphenous veins are particularly liable to thrombo-phlebitis, a condition to which varicose veins and strains of the foot, ankle and calf muscles may be predisposing factors. Long distance runners are very prone to varicose veins and therefore to thrombo-phlebitis; while it may also follow a strenuous cycling race.

Arterial Thrombosis

The initiating factors are injury of a vessel, increased coagulability of the blood and slowing of the blood stream, as in low blood pressure.

Volkmann's Ischaemic Contracture

This is an example of a complication which may be avoided by appropriate early treatment. In dislocation of the elbow and fractures of the forearm, embarrassment of the circulation must be carefully anticipated. If there is any sign of fascial tension causing pressure on the veins, it must be relieved by operation. A tourniquet should never be used if the forearm is much swollen: a compound wound must not be sewn up tightly. Any plaster-of-

Paris cast used for a damaged elbow or forearm must be padded, unless one side is split to allow for expansion of the tissues. Sometimes, however, the original injury may have caused so much internal bleeding and extravasation of traumatic swelling that the only certain way to prevent necrosis of the muscles of the forearm and hand characteristic of Volkmann's contracture, is immediate operative decompression. A similar necrosis of the extensor muscles of the anterior compartment of the leg may occur as the result of a direct kick over the front of the lower leg or from vascular disturbances caused by prolonged bicycling or long distance walking. Prompt operative relief of the tension may be the only method which is effective.

Unabsorbed haematoma. Complications occuring as the result of an untreated haematoma have already been described. (Chapter 2; Pathology: p. 274).

Fat emboli. These occur more often than is generally supposed. Fat particles released from fractured bones, particularly the femur, settle in the lungs. This complication can hardly be avoided.

Superficial sepsis. Contused, ragged skin wounds, if they are not properly cleaned, are very liable to low-grade infection. Such sepsis may later prevent or delay an essential operation on a deranged joint, for example the knee. All such wounds should be cleaned as early as possible under a general anaesthetic. Necrotic areas of skin should be excised and the wound sutured.

Remote complications

These may be classified as follows:—

(1) The contracted scar.
(2) Stiff joints.
(3) Loose unstable joints.
(4) Chronic traumatic myositis.
(5) Myositis ossificans.
(6) Chronic stenosing tenovaginitis.
(7) Certain indefinite conditions, possibly as the result of a neuro-vascular upset such as Sudek's Osteoporosis and Secretan's Traumatic Oedema.
(8) Degenerative changes in joints—(a) osteochondritis dessicans and (b) osteoarthritis.
(9) Miscellaneous group including certain collagen diseases such as Dupuytren's contracture; and remote nerve involvement, such as that of the ulnar nerve, following injuries to the medial condyle of the humerus many years previously.

1. *The contracted scar*

Conservative treatment: the full co-operation of the patient must be obtained for he can contribute greatly to the suppleness of the scar by sponging it with hot and cold contrast compresses, massaging the area with lanoline—starting at the periphery and working in to a central point, puckering the skin off deep structures and contracting the underlying muscles. His efforts may be supplemented by physiotherapy, which by stimulation of the underlying muscles, improves the circulation and makes the scar more supple. One sometimes sees old injuries with scar tissue adherent to the subcutaneous border of the tibia or one of the bony points of the elbow. The skin over the bone may be as thin as tissue paper; but it is possible by intensive treatment, which includes frequent injections of small amounts of 2 per cent procaine or xylocaine and hydrocortisone on to the edges of the scar, to render the skin more supple and non-adherent.

Operative measures: in some cases conservative treatment is too slow; it may take a year or even longer to produce only a moderate result. Plastic surgery lessens that period and gives a better final result.

Loss of whole thickness of the skin: when the patient is seen soon after injury, the wound is carefully cleaned and, if possible, whole thickness grafts applied. If there is any suspicion of sepsis it is better to use split skin postage stamp grafts. If some weeks have elapsed since the injury, treatment is similar to that recommended for the neglected contusion, except that if a skin graft is to be applied, it is best to use a whole thickness graft rather than a split skin graft or a Thiersch graft. This may mean the employment of a pedicle flap.

2. *Stiff joints*

If the original injury has been severe and the treatment inactive or inefficient, an injured joint may become painful and limited in movement. The presence of protective muscular spasm indicates an irritation of the joint due either to articular cartilage contusion, meniscus injury, adhesion formation limiting movements or even a loose body.

The muscles are usually found to be atonic and weak, and there may still be traumatic oedema in the tissues around the injured joint. Much of the original traumatic oedema may have been absorbed; but some may still remain, despite treatment, and some may have been organised into adhesions. The muscles lose not only power but also bulk; the circumference of the limb above the joint may be measurably less than that of the normal limb.

Though there may be adhesions limiting movement they should not, at this stage, be broken down by manipulation under an anaesthetic. The oedema must first be cleared away and the muscle tone improved by physio-

therapy and home treatment, as manipulation would cause a violent reaction and increased swelling. This has the effect of encouraging further adhesion formation and more oedema, so that a state is produced which is worse than before the manipulation.

Pain, swelling, loss of movement and muscle wasting often persist because the joint is not sufficiently protected and supported. When the joint is used, it is subjected to jars and strains. If it is properly protected and supported by a removable plaster case or a felt pad and bandage, the traumatic oedema around it will quickly disperse, for there is no longer a condition of strain.

At first the joint should be relieved of the strain of body weight by suitable support, and given remedial exercises by a carefully graduated course. The limb should be elevated for certain periods during the day in order to promote drainage of oedema fluid. At these times it is convenient to carry out any appropriate physiotherapy.

Gentle manipulative movements help to increase the range of movement slightly each day. If the range of movement is diminishing, the physical treatment, particularly the manipulations, may have been too vigorous. Another possibility is the influence of a septic focus somewhere. A time comes, however, when the oedema has subsided and the joint is still left with limited movement. Controlled manipulation under full anaesthesia is now indicated to break down adhesions. After this, home treatment, exercises, physiotherapy and further gentle manipulations must be given as often as possible. It is sometimes better to perform a series of graduated manipulations under anaesthesia, obtaining a limited increase in the range of movements each time, rather than run the risk of too great a reaction from too vigorous a manipulation.

The methods of manipulating joints have already been discussed. The technique is similar whether an anaesthetic is used or not. An injection of hydrocortisone with hyaluronidase may help to reduce any reaction to the manipulation and is given while the patient is still anaesthetised.

In certain chronic injuries of the knee joint, particularly those to the medial collateral ligament and lesions accompanied by haemorrhagic synovitis, gentle progressive home treatment and physiotherapy may have to continue for three months before the time comes to subject the joint to a manipulation under anaesthesia. Provided a small degree of improvement in the range of movement is being obtained each week, it is better to continue with general conservative methods. When improvement has stopped, controlled manipulation under general anaesthesia by breaking down adhesions, often produces a definite increase of the range of movement. Traumatic capsulitis of the

shoulder joint is another condition which responds well to this type of management.

It is sometimes difficult to decide whether to rest a joint or to start manipulations and vigorous treatment. If there is any doubt, it is better to treat the case by resting it from all forms of strain such as weight bearing, and by some form of removable support which can be taken off at intervals during the day for exercises and physiotherapy, including gentle manipulations. In a patient over sixty, manipulations under anaesthesia should be carried out only with great caution. It must be remembered that bones are inclined to be brittle at that age and the soft tissues, having lost much of their flexibility, oppose the restitution of full free movements.

If it is absolutely necessary to manipulate an elderly patient under an anaesthetic, it is wise to give an extensive course of preliminary physical treatment first, so that the manipulation simply breaks down terminal adhesions with only slight reaction.

3. *Loose, unstable joints*

Joints may be unstable for the following reasons:—

(a) wasting of muscles such as the vastus medialis when the knee has been injured. The training of the static prime fixing muscles at all levels is of paramount importance.

(b) laxity or rupture of ligaments due to injury,

(c) osteoarthritis, with gradual disintegration of the joint, and

(d) neurotrophic condition, such as Charcot's joint.

Naturally the primary cause must be considered and treated; but suitable protection and support of the joint in a plaster cast may be the first consideration. The joint may need support for one to three months during which time the patient carries out graduated isometric contractions against the resistance of the cast. When the lumbo-sacral spinal joints need the support of a plaster jacket, it is a wise principle to leave on the plaster cast for a month after all symptoms have disappeared. If a closed cast is not considered necessary, a split one has the advantage that it can be taken off for home treatment and physiotherapy, in which faradic contractions are particularly important. The active approach, in which Active Alerted Posture is the most important factor, will help control joint movement and produce joint stability.

Operations on loose joints include:—

(i) Reinforcement of a torn ligament by a fascial graft.

(ii) Repair of a torn ligament.

(iii) The transfer of a tendon to take the place of a torn ligament.

(iv) Arthrodesis of a painful unstable joint when other methods of treatment have failed.

4. *Chronic traumatic myositis*

Muscles are frequently shortened as the result either of direct or indirect trauma. The extensibility of the muscle is diminished by the formation of tender thickenings, scar tissues or adhesions in the muscle itself. It may become adherent to neighbouring structures. True traumatic fibrosis of the muscle occurs as the result of the organisation of traumatic effusion.

It has been suggested that chronic postural strain can cause a specific lesion in muscles. This begins as tenderness of the anti-gravity or postural muscles. If the strain is allowed to continue the underlying joint eventually becomes strained and surrounding muscle spasm develops. This spasm, unless it is relieved, results in irreversible shortening of the muscles, due to interstitial fibrosis as occurs in established osteoarthritis. Postural training and Active Alerted Posture are the best means to prevent these changes. If they have already begun, they may be checked by an active approach to posture and treatment which includes regular physiotherapy.

Before the surgeon considers any operative treatment he ought first to try conservative measures—home treatment, physiotherapy with gentle manipulation and injections of hydrocortisone. If the patient stops improving at any stage manipulation under pentothal may be attempted.

The manipulation of the constricted muscles is carried out in the way already described, particular attention being given to manipulating the joint above and below the site of injury.

In certain muscle injuries it is possible to obtain quicker results by operative measures, especially in the young patient with muscle ruptures or partial ruptures of the quadriceps and abdominal obliques. In chronic muscle injury, however, conservative treatment often obtains a cure and should include physiotherapy, injections, gentle manipulations and graduated exercises. These should be given three weeks' trial before a controlled manipulation under pentothal is attempted.

However, these measures sometimes fail and examination of the muscle by open operation may be indicated. In these cases tenotomy of the origin or insertion of the muscle may be necessary to allow it to seep away from the attachment. This appears to prevent chronic strain with reactionary fibrosis. Occasionally the tendon of a contracted muscle may have to be lengthened.

After operation it is important to prevent further strain by carefully grading the exercises and the physiotherapy.

5. *Myositis ossificans*

This condition often occurs following a dislocation especially of the elbow joint. Structures often involved are the capsule on the inner and outer aspects of the joint or the tendon of insertion of brachialis anticus in front. Some authorities believe that provided movement is not excessive, it has no effect on the amount of calcification. A haematoma forms at the site of injury and this with the contusion of the muscle forms an avascular inert mass which becomes calcified. Therefore care and gentleness must be taken in the reduction of the dislocation.

At the shoulder joint the supraspinatus tendon or the subacromial bursa is frequently subject to calcareous changes. Both of these latter conditions frequently follow an external rotator cuff injury. The tendon and the bursa may become involved in calcareous changes (as occurs in the brachialis anticus) but sometimes it may be a more gradual process partly due to chronic strain affecting the blood supply through the paratenon and partly due to attrition or rubbing of the tendon and bursa on abduction of the arm. In treating injuries to the elbow joint and the shoulder joint it would seem that the early dispersal of traumatic effusion by a combination of rest from strain, gentle exercise avoiding strain, home treatment including hot baths, contrast baths, home massage and exercises with supervised physiotherapy is an essential factor in preventing these complications. Forcible manipulation is to be condemned as adhesions of the muscle around the site will be torn and a reaction set up with further extension of the myositis.

When the thigh muscles are involved in an established myositis ossificans, treatment of the structures around the knee and hip joint is imperative. Some of the original traumatic effusion is bound to have seeped down the thigh and caused reaction, even involving the structures of the knee joint itself. By judicious exercise without strain, appropriate home treatment and physiotherapy, including gentle manipulation and injections, the condition can usually be improved.

6. *Chronic traumatic teno-synovitis*

In spite of active treatment in a recent injury, adhesions may form between the tendon and its sheath. They may resist local injections of procaine, hydrocortisone and hyaluronidase, and operation to free the tendon from its sheath may become necessary. However, conservative treatment should al-

ways be given a chance, even when the teno-synovitis involves the flexor hallucis longus tendon at the under surface of the metatarso-phalangeal joint where it passes between the two sesamoids. This condition is common in athletes such as fast bowlers and high jumpers; it is due to the jarring and contusion of the tendon when the foot hits the ground.

7. *The effects of vascular disturbances*

If a vessel of any size is injured, blood escapes into the tissues and as it clots, forms a haematoma. We have already stressed how important it is to distinguish between true venous thrombosis and the clotting of a haematoma. The former, often due to thrombo-phlebitis, can lead to serious complications such as a pulmonary embolus; the latter is a normal physiological process. However, active measures to disperse the haematoma either by expressing it through an incision or inducing it to absorb by active treatment and injection are always necessary and may avoid later complications. Women who develop 'white leg' in pregnancy may suffer from persistent swelling and oedema with pain and loss of function. The patient is sometimes given the impression that no form of treatment is likely to be of benefit, but much can be done by appropriate treatment—postural drainage, massage, faradism, remedial exercises and bandaging. One must not expect an immediate result. It may be necessary to persevere for months before the leg begins to improve and then home treatment must be continued indefinitely in order to avoid a return of the symptoms. A former athlete who was a lady doctor came for a consultation in 1948. She had developed 'white leg' on the left side in a confinement twenty years previously and in the right leg in another confinement twelve years before. Both legs swelled and ached, and these symptoms caused her to give up all forms of sport, because exercise accentuated the symptoms. With the legs elevated, massage and faradism under pressure was given three times weekly for six months and together with home treatment, she made an excellent recovery and was discharged to continue home treatment. In 1955 she had a slight return of symptoms but these have been kept under control by monthly treatments of physiotherapy together with her conscientious home treatment.

8a. *Osteochondritis dissecans*

It is well known that the toughness and consistency of articular cartilages vary in individuals. A single area of osteochrondritis dissecans may, however, occur following acute trauma. Possibly in these cases injury has produced a subchondral haematoma and the overlying articular cartilage separates as a loose body. Smillie and Pridie have shown cases in which it has been possible

to replace the piece of separating cartilage, and the joint surface has been almost restored to normal.

Operation is best delayed until the separation of a fragment of cartilage is about to occur and is inevitable.

8b. *Osteoarthritis*

This is such a common complication of injury in the athlete that its course and treatment are given in some detail.

There are probably several factors occurring together which account for the onset and production of osteoarthritis. The process is said to commence in the articular cartilages but if operation is performed in early cases the synovial membrane is thickened and greyish yellow in appearance. Trueta[1] describes a hyperaemia of the blood vessels but whether this is a primary or secondary phenomenon is open to discussion. In all probability it is secondary, and is an attempt by the patient's protective reaction to try and absorb the products of joint degeneration. Trueta[2] has also shown that articular cartilage varies in texture at different sites even on a weight-bearing surface like the acetabulum. In this cavity the main weight-bearing area is the superior central part and here the cartilage has a preponderance of chondroitin sulphuric acid, which makes for toughness. This type of cartilage should predominate in the articular cartilage of all weight-bearing areas in distinction to that in the non-weight-bearing areas, in which there is an excess of collagen fibres.

Faulty posture, especially if it is a habit of years, contributes to the onset of the condition because constant strain is thrown on areas of a joint not meant to take the main stress, areas in which the articular cartilage is relatively soft. When, for example, there is a slight eversion of the foot of a patient standing 'flatfooted', valgus strain is thrown on the inner side of the knee joint and thigh. The head of the femur is slightly rotated outwards, so it does not make contact with the true weight-bearing surface of the acetabulum. This slight strain, working over many years, may be one of the factors in the production of osteoarthritis in both the knee and the hip joints.

In the early phase, because the condition is confined to articular cartilage, the degeneration may be reversible; early and prolonged treatment may produce a symptom free joint, provided the patients continue home treatment indefinitely. Frequently the early osteoarthritic joint is completely symptom-free, until symptoms are started by prolonged over-use of the joint, in which case the accumulative effects of wear and tear have reached a threshold; or by a relatively trivial injury, in which adhesions are stretched; or by the separation of a small fragment of articular cartilage which causes the joint to catch

without locking. Acute signs and symptoms may persist until the denuded area has healed over and the piece of cartilage becomes enveloped and surrounded by a synovial fringe or gradually disintegrates in the synovial fluid.

Through the work of Smith Peterson and recently that of Trueta[3] and Lloyd Roberts[4] it has been shown that articular cartilage can regenerate. The medical profession generally would have been sceptical about this thirty years ago. Osteoarthritis of the ankle, knee and hip joints follows on injuries and fractures of these weight-bearing joints so frequently in later life that endeavours should always be made to evolve some routine of home treatment which might make the patient less disposed to a condition which otherwise seems almost inevitable. Every injured athlete should be warned of this complication by his doctor; and he should make every effort to regain and maintain full range of movement in the joints associated with an injury. The muscles around an injured joint are frequently never restored to full tone and power after an accident, either because the importance of full recovery is not appreciated, or because the patient will not make the necessary effort. Such lack of appreciation may account for the appearance of osteoarthritis in the hip following a low back episode (due to disc degeneration) in which there has been a severe sciatica, and the buttock and thigh muscles have been left wasted. Frequently surgeons see joints, injured many years previously, the controlling muscles of which are wasted and atonic and in which there is loss of the extremes of joint movement. Such joints may be improved by an enthusiastic course of home treatment and perhaps physiotherapy; and if osteoarthritis has not already shown itself, its progress can be delayed or checked. A patient who has had an injury to a joint should be told to report as soon as symptoms of pain and stiffness appear so that the correct treatment may be prescribed.

The treatment of osteoarthritis may be summarised under the following headings:—

(i) Preventive treatment.
(ii) General treatment.
(iii) Local treatment.

(i) *Preventive treatment.* The substitution of Active Alerted Posture for Inactive Slumping Posture is essential. By adopting Active Alerted Posture as often as possible the patient takes the strain off weight-bearing joints and so reduces the chronic trauma which leads to osteoarthritis.

The reduction of weight by dieting is equally important. The overweight patient is putting extra strain on his weight-bearing joints and in time this encourages the development of osteoarthritis.

Simple rituals of movement in everyday life will help to preserve the full range of joint movements and power in the muscles acting on the joints. For example, one should always rise from a chair by using the thigh muscles and not by pressing on the arms of the chair with the hands (in which case it is largely the triceps muscles on the back of the arms which raise one to the erect position and not the hip and thigh muscles). Stairs should be mounted two or three at a time to ensure a full use of hip flexion and extension. Long car rides should always be followed by a few seconds of stretching and full knees-bend movements. In these and many other little ways the body can be kept strong and supple and the onset of degenerative changes delayed. Lifelong health and suppleness is not a birthright, it is a way of life.

Joints previously damaged must be cared for. The damage may be due to injury or to a lesion elsewhere affecting the muscles round the joint—for example the wasting associated with brachial neuritis or sciatica as a result of cervical or lumbar disc lesions. Every joint injured throughout life is a potential source of osteoarthritis and therefore the patient should be warned and make every endeavour to keep the full movements of the joint, as well as full power in the muscle for the rest of his life.

(ii) *General treatment*. Septic or toxic foci should be eliminated. A suitable balance of rest and exercise should be prescribed and joints relieved of strain by the use of sticks, crutches or other forms of suitable support. Pain should be controlled by analgesics.

(iii) *Local treatment* is divided into five stages, three of which are conservative, the fourth includes minor operations and the fifth major operations. It is impossible to tell from the X-rays of an osteoarthritic joint exactly how severe the symptoms are. However, it is usual for those who have marked symptoms to have advanced radiographic changes, although this is not always the rule. Some patients with only early changes may have severe symptoms, which are probably due to erosion of the articular cartilage. However, even the severely crippled cases often obtain benefit if they are given the whole range of conservative treatment outlined in the first three stages.

The First Stage: This consists of home treatment with periodic courses of physiotherapy. The patient must be prepared to carry out the home treatment conscientiously twice daily. The measures are simple ones such as self-massage and exercises following contrast baths in the morning and hot baths in the evening. If possible he should have formal physiotherapy twice, or at least once, a week, short wave, massage and manipulation, or massage, faradism and manipulation.

The Second Stage is similar to the first, except that to home treatment and physiotherapy are added periodic injections of hydrocortisone which is given intra-articularly.

The Third Stage. If the patient still has symptoms, he should go on to the third stage in which the treatment is the same as that of the first stage with added intra-osseous injections. If the knee joint is affected, for example, the injections are made into the marrow cavity of the lower end of the femur and the upper end of the tibia. Bone marrow is removed by aspiration and Lawton Scott's solution put back in its stead. This solution consists of :— Acid salicyl 0·2 per cent, sod. citrat, 1·0 per cent, chlorbutol 0·5 per cent, phenol. red 0·2 per cent, aqua. pro inject, to 100 ml. In this stage, also, one may include manipulations under pentothal and deep X-ray therapy.

The Fourth Stage. If conservative treatment fails the surgeon may advance to the fourth stage of minor operations. These consist of tenotomy, mylotomy, local arthrotomy or joint irradiation by cobalt 60 needles.

The Fifth Stage. The final stage, if the patient still has much pain and limitation of movement, is that of major operation. We may illustrate these by listing the operations available for the hip:—

(1) Arthroplasty: (a) Smith Petersen cup arthroplasty.
(b) Austin Moore or Thompson metal prothesis arthroplasty.
(c) Charnley's low friction cup arthroplasty.

(2) Arthrodesis.

(3) Adjustment of lines of strain as in MacMurray's displacement osteotomy of the femur.

(4) Removal of the head of the femur—Girldestone's operation or Batchelor's pseudarthrosis.

(5) Operation on nerves, spinal cord, or brain to remove pain. This form of therapy is now seldom employed.

Even after a successful operation, home treatment must never be given up. The corresponding joint on the opposite side, and those above and below, must be included; when the arthritis is in the right hip, the right knee, the low back and the left hip must all be included in the daily routine of massage, exercises and baths in the home.

By continuous conscientious home treatment combined with physiotherapy, the moderately severe osteoarthritic patient may be kept relatively symptom-free for years. If minor or major operations have to be resorted to, they are made more effective by the improvement in the muscle tone and power and increased joint movement.

The osteoarthritic must realise that there is no absolute cure for his condition and that he must continue with home treatment and periodic courses of physiotherapy indefinitely.

9. *Miscellaneous group*

There are a large number of conditions which appear to be initiated or aggravated by injury. Examples are the fibrotic changes of Dupuytren's contracture which occurs in the palmar fascia of elderly athletes following a Colles' fracture; in the transverse carpal ligament of the wrist leading to median nerve compression in the carpal tunnel; and in the flexor tendons of the fingers (stenosing tenovaginitis). With appropriate treatment directed to the Colles' fracture and physiotherapy directed to the palmar fascia, annular ligament and tendon sheaths, these complications can often be forestalled and avoided.

Collagen diseases so often follow injuries to the middle aged athlete that the surgeon should always be on his guard, ready to give the physical treatment that can abort the disease and prevent the disability.

Section II Fractures in Athletes

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1

THE PRODUCTION AND HEALING OF FRACTURES

THERE is no essential difference between fractures occurring in the course of games or sport and those produced in any other way. They are caused by the same types of mechanical force, the same principles of treatment apply to their management, union takes place in the same way and in the same time, and the end results are determined by the same factors. Indeed, the majority of bony injuries in athletes are indistinguishable from those sustained in the home, at work or in going from place to place in the ordinary way.

There are, however, some special features associated with fractures in athletes which may influence their treatment. In general such injuries occur in young and healthy adults and the problems peculiar to fractures in infancy, childhood and old age do not therefore arise. The precise end-results too, are of more significance than might normally be the case, a trivial permanent disability of no great importance in ordinary life might end the career of a first class athlete. Finally the time factor may be of even greater importance than usual; for example, a long period of disability or partial disability might deprive a professional athlete of his means of livelihood. These special circumstances must be borne in mind by those responsible for the management of sporting injuries and may determine the methods of treatment employed.

The treatment of all fractures is based on certain principles which are of themselves relatively simple. These principles, in turn, depend on the way in which bone and the surrounding tissues react to injury and the immediate and late effects of this reaction. The basic principles may be commonplace to orthopaedic surgeons, but far too often they are overlooked in the welter of technical detail associated with the management of different types of fractures. Since clear understanding of basic principle is essential to all those engaged in any part of the management of fractures, this must be dealt with before the individual fractures are considered on a regional basis.

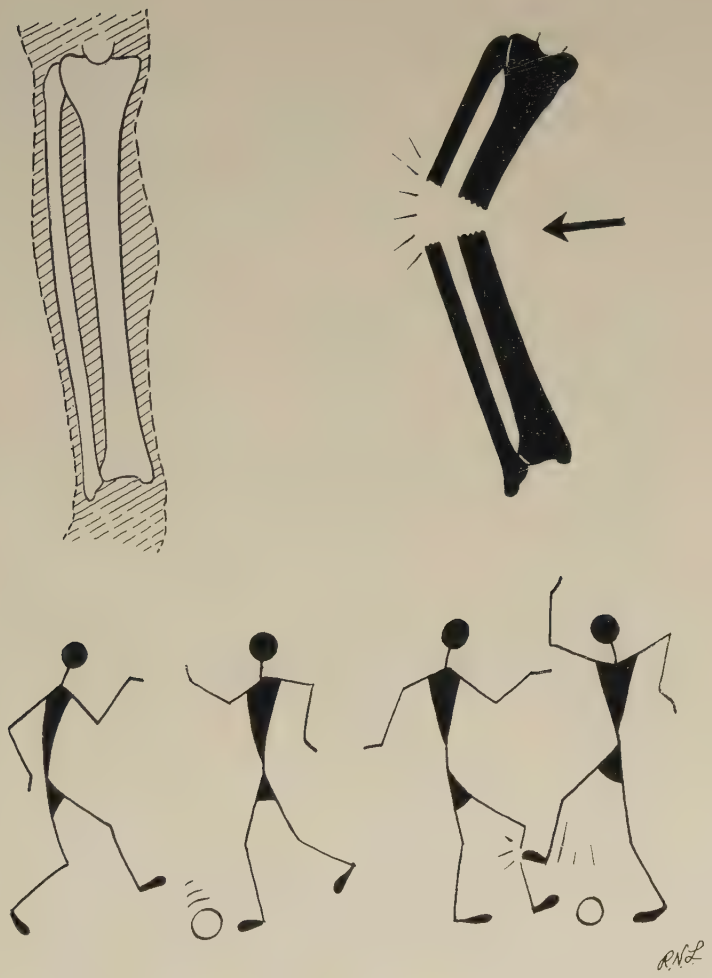

FIG. 1. *Fractures produced by direct violence.* Force falls directly on the bone in a plane across its long axis, producing a transverse fracture at the point of application of the force. In sport such force is usually due to an outside agency, something moving comes into contact with the moving or stationary object or the moving object comes into unexpected contact with a stationary object. The bones of the forearm and of the leg below the knee are particularly exposed to direct violence.

THE PRODUCTION OF FRACTURES

The most essential property of bone is its ability to transmit force of all kinds constantly or intermittently without being distorted or damaged. The skeletal system as a whole has many functions. It contributes rigidity to the body, supplies sites for the origin and attachment of muscles, provides levers and fulcrums for the production of movement and forms protective coverings for vital organs: and all these functions depend on this faculty to transmit force. Further, not only is normal bone not harmed by the stresses which it has to bear, but it actually hypertrophies and becomes stronger in response to these stresses. This phenomenon is not shared by the only other two structures in the body, articular cartilage and the intervertebral discs, which are concerned in the constant transmission of force.

There is, however, a limit to the amount of force which an individual bone can stand; and when that limit is exceeded the bone breaks, that is, it is fractured. The nature of any fracture depends not only on the size and shape of the bone involved but on the type of force by which it is produced. Broadly speaking there are five ways in which mechanical forces too great to

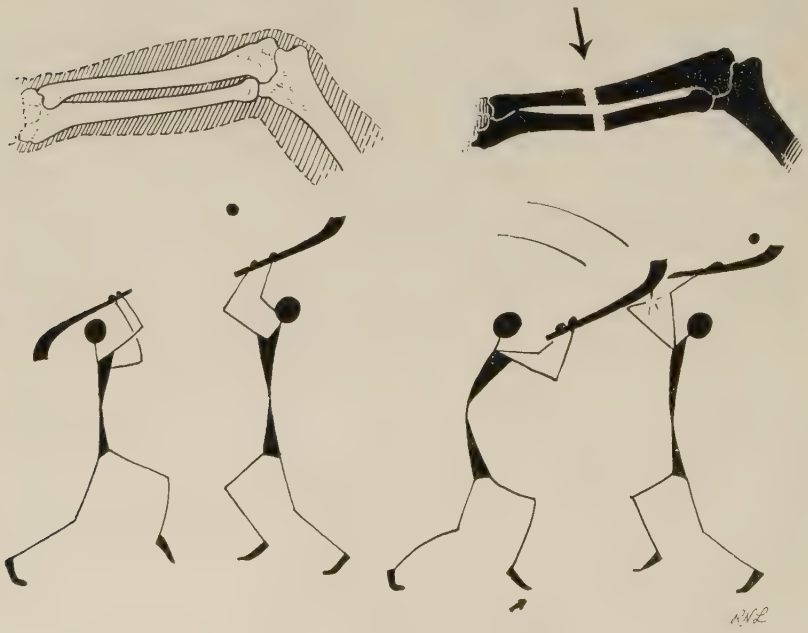

FIG. 2. See Fig. 1 overleaf

bear may be applied to bones, and the fractures produced in these different ways have certain characteristic features.

The simplest type of force is one falling directly on a bone in a plane across its long axis, that is in the plane in which the bone is weakest. If such a force is of sufficient violence the bone breaks transversely at the point at which the force is applied (*Figure* 1). In sport such a force is usually due to an outside agency: something moving comes into contact with the moving or stationary body or the moving body comes into unexpected contact with a stationary object (*Figure* 2). The degree of violence affects the nature of the fracture

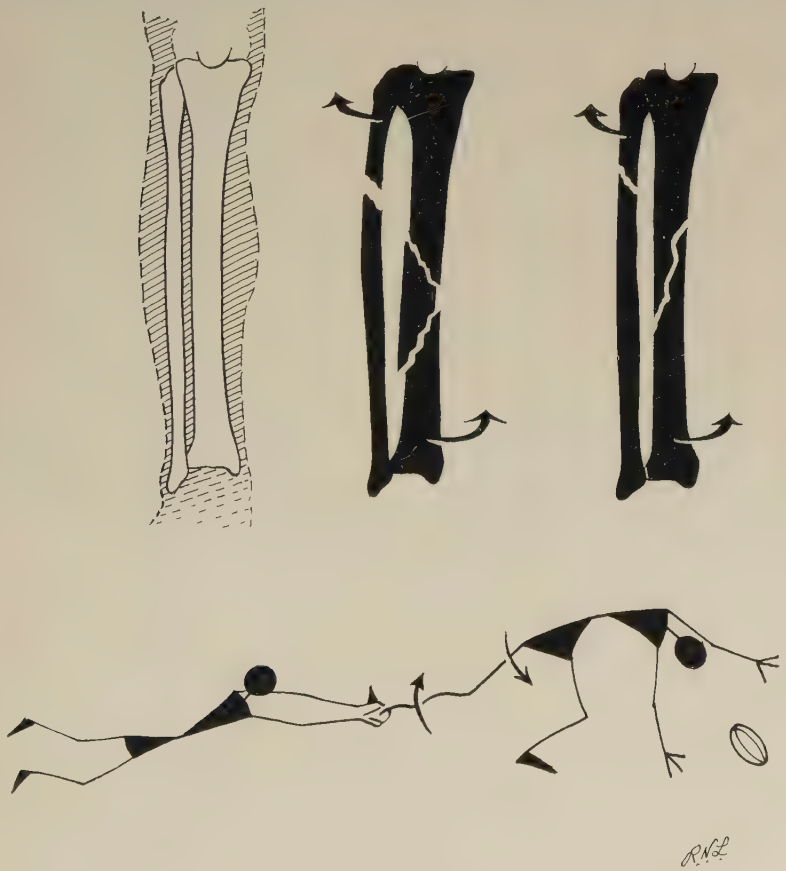

FIG. 3. *Fractures produced by indirect violence.* The ends of a limb are subjected to rotatory forces tending to move them in opposite directions. The bone breaks obliquely at its weakest point, and the fractures so produced may be single or multiple.

produced. Ordinarily the affected bone or bones break transversely at the point of impact, but if the force is sufficiently great, the bone is actually shattered into fragments at this point, the so-called comminuted fracture.

Less simply but probably more commonly, violence applied indirectly produces a fracture. This occurs when the extremities of a bone are subjected to forces, usually of a rotary nature, which tend to move them in opposite directions. If these forces are sufficient the bone breaks at its weakest point and the line of fracture is oblique in relation to its long axis (*Figure* 3). In sport the force producing such fractures, which are relatively common, is usually body momentum or body momentum combined with an outside agency (*Figure* 3).

The third type of force which may produce a fracture is transmitted along or through the bone itself, that is a force applied at one end or on one surface of a bone which tends to compress it as a whole (*Figure* 4). Such a force may crush or compress the entire bone or, in a long bone, break off part of one extremity. The compression fractures are most commonly produced by an abrupt checking of body momentum. When such a check is anticipated,

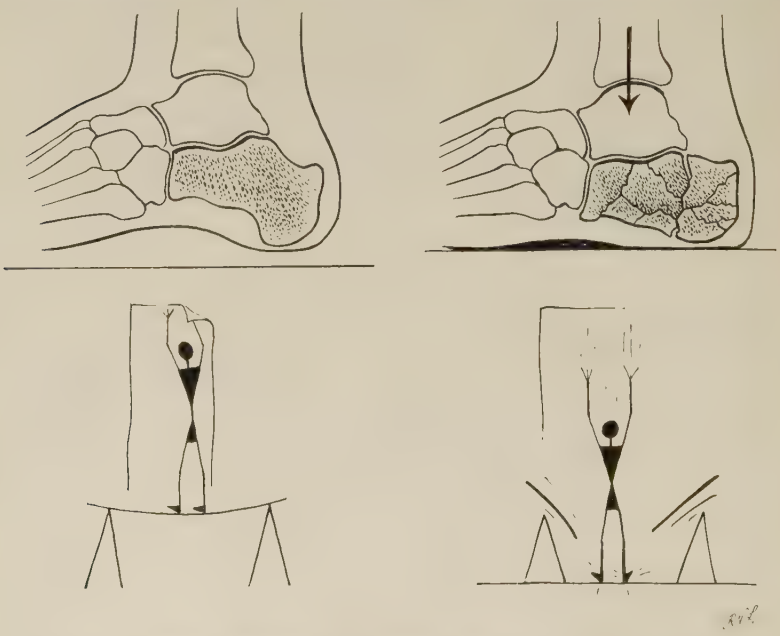

FIG. 4. *Fractures produced by transmitted force.* Force transmitted through the bone itself may tend to compress it as a whole, producing a comminuted type of fracture.

bony injury can be prevented if the body is held in a position of flexion so that the force is absorbed by the elastic muscles. For this reason such fractures are unusual in trained athletes.

Force sufficient to cause a fracture may be transmitted to bone through a muscle, a fascial plane or a fibrous ligament. Such a force is generated either by violent muscle contraction or by an abnormal range of movement and is always of a traction, that is of a pulling, nature. The fracture produced consists of the tearing off of a fragment of bone in the region of the attachment of the structure through which the pull is transmitted (*Figure* 5). A variety of this type of force can produce complete fractures of the patella.

Finally, certain special types of fractures, the so-called 'stress' or 'fatigue' fractures, occur as the result of the constant repetition of forces or stresses not in themselves sufficient to damage normal bone. These fractures, which have many special features, are considered in a separate section.

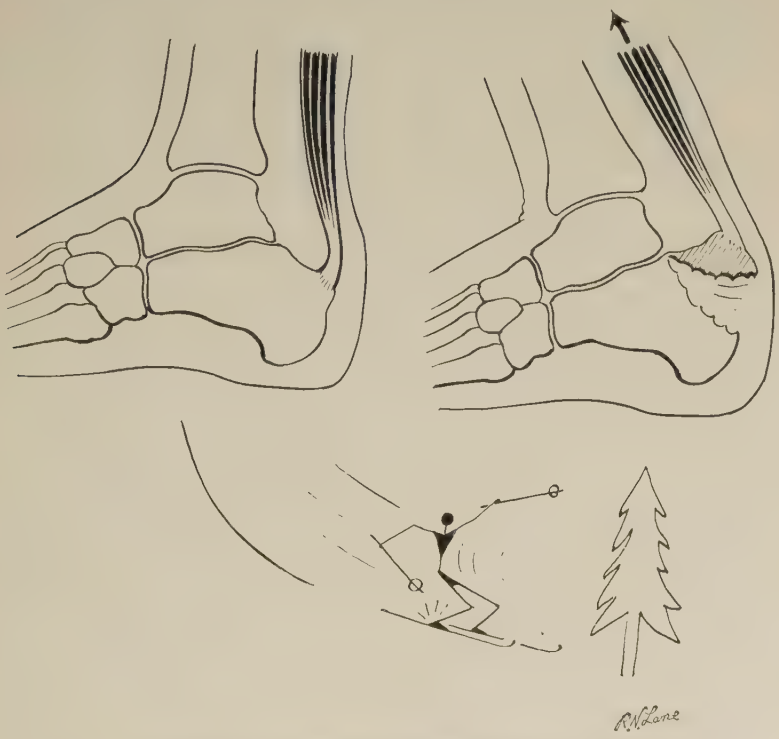

FIG. 5. *Fractures by force transmitted through muscle or tendon.* A sudden contraction of a powerful muscle may tear off part of the bone at the point of attachment of this muscle.

HEALING OF FRACTURES

Natural healing. When a bone is damaged a process of natural repair or healing commences and, in favourable circumstances, the continuity and strength of the bone is ultimately restored, that is the fracture unites. This process of union is an extension of the changes whereby any damaged body tissue is repaired by the formation of fibrous tissue; but the conditions necessary for the repair of a fracture by solid bone are more exacting than is usually the case with soft tissue. One of the primary aims of treatment of all fractures is, therefore, the establishment of the best possible conditions for natural healing.

When a bone is broken bleeding occurs both from the bone and from adjacent tissue, and collections of blood quickly form around the fracture surfaces. (*Figure* 6A). Normally these collections merge to form one single haematoma surrounding the whole of the damaged area (*Figure* 6B). This enveloping haematoma, forming soon after injury, is an essential feature of union, and within it further repair takes place. In the early stages the outer layers of the haematoma clot and become solid while its interior remains fluid, being cut off from the general circulation (*Figure* 6C).

The next stage is the replacement of the interior of the haematoma by granulation tissue. Capillary loops grow from the bone ends, invading the haematoma and ultimately uniting so that they bridge the gap between the fracture surfaces (*Figure* 6D).

The highly vascular and extremely friable tissue so formed is then gradually replaced by soft fibrous tissue, laid down by fibroblastic cells which arrange themselves parallel to the capillary walls. As this fibrous tissue is produced, the capillary loops wither away and the whole area becomes less vascular and a little less easily damaged (*Figure* 6E).

The fibrous tissue is then gradually infiltrated by cartilage which is deposited among the fibrous strands and ultimately replaces it altogether. This cartilage, at first soft and semi-elastic, becomes harder and more rigid, ultimately forming a bony mass, the callus. When first formed this callus constitutes an irregular mass surrounding and joining the fracture surfaces, invading the medullary cavity if it has been exposed and bridging the gap between the bone ends (*Figure* 6F). Ultimately the callus becomes sufficiently strong and rigid to restore the continuity and strength of the bone as a whole.

When this stage has been reached the way is clear for the final process of repair, a re-moulding of the repaired area. Excess callus is absorbed both from the outside and from the area of the medullary cavity, the internal structure of

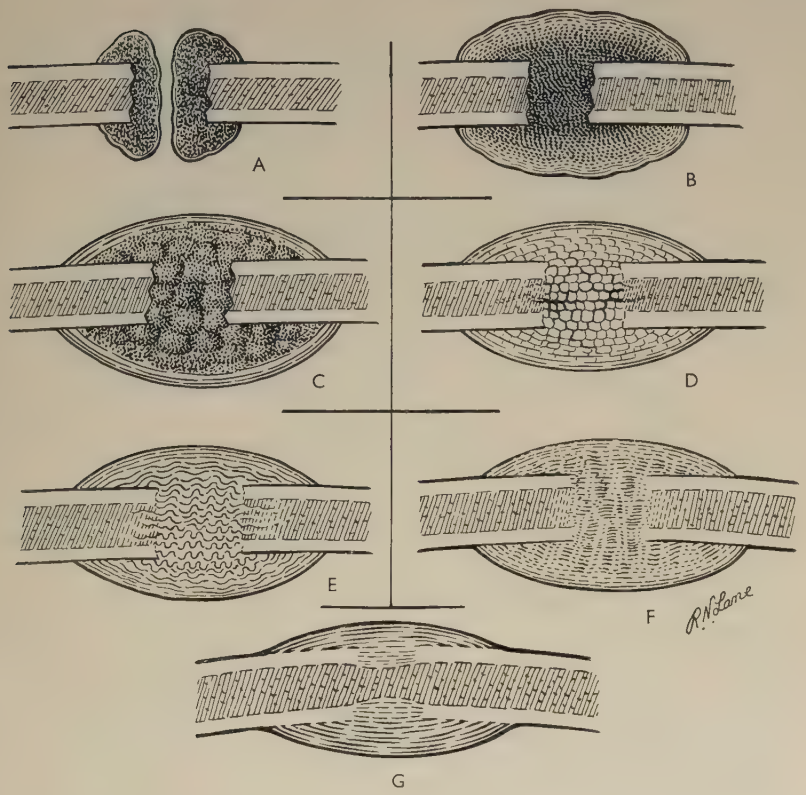

FIG. 6. *Healing of a fracture.* When a bone is broken the ends bleed (A). Normally the haematomata so formed merge to surround the damaged area (B). In the early stages the outer layers clot while the interior remains fluid, being cut off from the general circulation (C). The fluid interior is then replaced by granulation tissue consisting of capillary loops which grow from the bone ends (D). The soft granulation tissue is next replaced by fibrous tissue, strands being laid down parallel to the capillary walls (E). The fibrous tissue is then infiltrated by cartilage which is deposited among these strands and gradually becomes harder and more rigid to form a mass of undifferentiated bone, callus (F). Finally after the callus has become consolidated excess bone is absorbed from the outer sides and the medullary cavity, restoring the shape and structure of the damaged bone almost to normal (G).

the remaining new bone is re-organized in accordance with the stresses to which it is subjected, and finally the shape and structure of the damaged bone is restored almost to normal (*Figure* 6G).

This process of union is accompanied by various biochemical changes within the fracture haematoma and by the migration to the area of those body cells which produce fibrous tissue, cartilage and bone. There is, however, no change in the composition of the blood as a whole nor can healing be speeded up by the introduction of any substance into the blood stream.

The time factor in the healing of fractures is fairly constant. Union occurs more rapidly in growing bones, that is in children, and broadly speaking the younger the child the more rapidly the fracture will unite. Once growth has ceased, that is in adults, the rate of union is fairly constant. Provided treatment is adequate in a healthy adult the time taken by a fracture to unite is determined by its site and the amount of bony damage to be repaired and the average time in similar fractures adequately treated does not vary very greatly.

THE FACTORS WHICH INFLUENCE THE HEALING OF FRACTURES

When a bone has been broken its subsequent healing depends on a number of different factors. In favourable circumstances the damage is repaired and the bone unites solidly; in less favourable circumstances union may be delayed or imperfect; and if conditions are unfavourable the fracture may never unite at all. Most, although not all, of the factors which determine how fast and how solidly a broken bone unites are under the control of the surgeon and a large part of the treatment of fractures consists of the establishment of the most favourable conditions possible for natural repair and the elimination of anything which might adversely affect this process.

The following are the more important factors which influence the healing of bone.

1. *Blood supply*. The natural repair of the fracture in all its stages is dependent on an adequate blood supply to the area. The initial phase, the envelopment of the whole damaged area in a haematoma, occurs as the result of bleeding from the fracture surfaces. If one of the main fragments of a broken bone has no blood supply the formation of this haematoma may be delayed or may not occur (*Figure* 7). Similarly, the second stage of healing, the bridging of the defect by granulation tissue consisting of multiple loops of capillary vessels is dependent on the presence of a free blood supply to the bone ends. This stage is greatly delayed if one main fragment is avascular, and will not occur at all if both have an inadequate blood supply. Subsequently the cells which produce the fibrous tissue, cartilage and bone essential to the later stages of healing must reach the newly forming tissue by way of the blood stream and unless these cells have free access to the entire area strong bone cannot be formed. At all stages, therefore, a good blood supply is essential

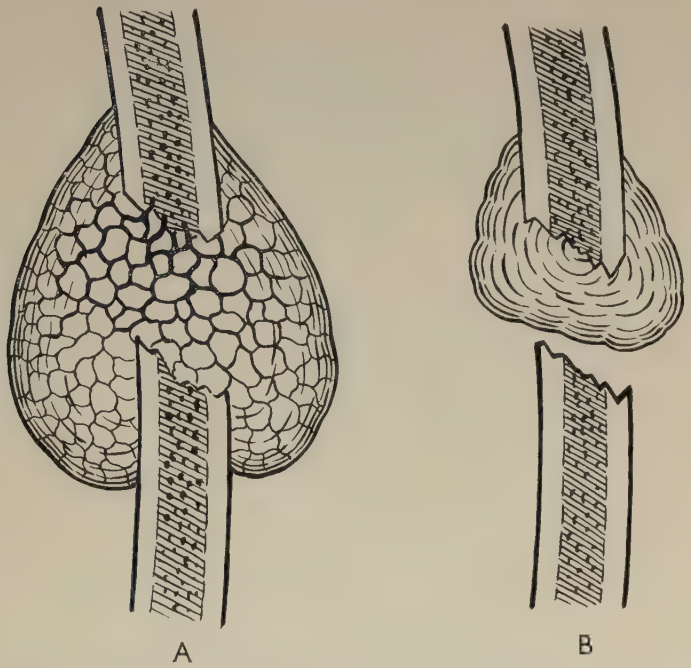

FIG. 7. *Influence of blood supply.* If one fragment of a broken bone has no blood supply a single haematoma is formed. In favourable circumstances this may envelop both broken ends (A), when union can take place, or it may fail to do so (B), when union is impossible.

and any vascular deficiency must be associated with delay or failure of union.

The position of completely isolated bony fragments such as may occur in comminuted or double fractures must be considered. Often, particularly in the case of smaller pieces, such fragments are cut off from any blood supply. Larger fragments may retain their blood vessels although this is not always so. Vascular fragments contribute to healing in the normal way, (*Figure* 8 A and B) but this cannot happen with pieces of bone which have no blood supply. Such fragments must themselves be incorporated in the haematoma, invaded by capillaries and replaced by living bone (*Figure* 8 C and D). These so called 'free' fragments play an invaluable part in healing, however, in that they form a scaffolding by or from which new bone is formed to bridge defects which otherwise might delay or prevent union.

The blood supply to the site of the fracture is not, of course, under the

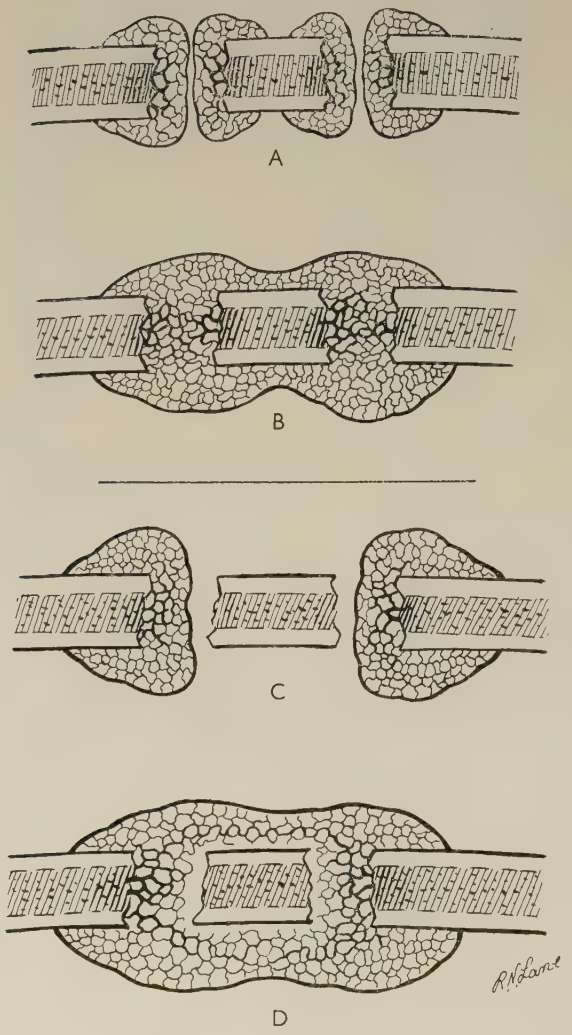

FIG. 8. *The influence of blood supply in comminuted fractures.* If all the bone fragments have an ample blood supply haematomata form at each fracture surface (A) and these readily merge to envelop the whole of the damaged area (B). If one fragment is avascular it forms no haematomata (C) and must be enveloped by those from adjacent fragments if healing is to become possible (D).

direct control of the surgeon. Bones vary greatly in their vascularity and different parts of long bones have different blood supplies. Fractures occur equally readily in all types of bone and the fracture itself may well damage the existing blood vessels. All that the surgeon can do is to make sure that once a fracture has occurred no measures are adopted which may further damage the surviving blood supply.

2. *The relationship between the bone ends.* The same forces which may break a bone also tend to separate the bony fragments after the fracture has occurred. The amount of separation produced depends both on the violence of the forces involved and on how long they are maintained.

The degree of displacement which occurs is very important for two reasons. From a long term point of view the restoration of normal function will depend on the continuity of the broken bone being re-established so that its fragments bear a normal relationship to each other and this aspect will be discussed in a later section. The exact relationship between the fracture surfaces has also an important effect on all phases of healing. The formation of the initial enveloping haematoma is obviously much simpler if the fracture surfaces are in contact or close together, and becomes more difficult if the bone ends are actually separated. If the separation is sufficiently great, or if soft tissue has become interposed between the bone ends, individual haematomata form around the fracture surfaces and further healing cannot take place. (*Figure* 9.) Again, once an enveloping haematoma has formed, the amount of subsequent repair necessary depends on the relationship between the bone ends. If these are in close and accurate contact repair is at a minimum, if they are separated the defect must be bridged and the more repair that is necessary the longer will the fracture take to unite.

Two other aspects of the relationship between the fracture surfaces have special significance in relation to healing.

After a bone has been broken the force to which the fragments are subjected are usually produced by muscle tone and are of a compressing nature, that is they tend to force the bone ends together or to overlap. Certain fractures of the long bones are treated by traction, that is by a constant force pulling in the long axis of the bone in opposition to the compressing forces of muscle tone. (*Figure* 10). If the traction force is excessive the bone ends may actually be pulled apart. This distraction has a very adverse effect on the healing of the fracture. The fragile capillary loops formed in the early stages are torn, and subsequently the fibrous tissue is stretched and under constant tension. This strangles the blood supply to the fibrous tissue and further healing is indefinitely delayed. Once distraction has occurred in this way, a

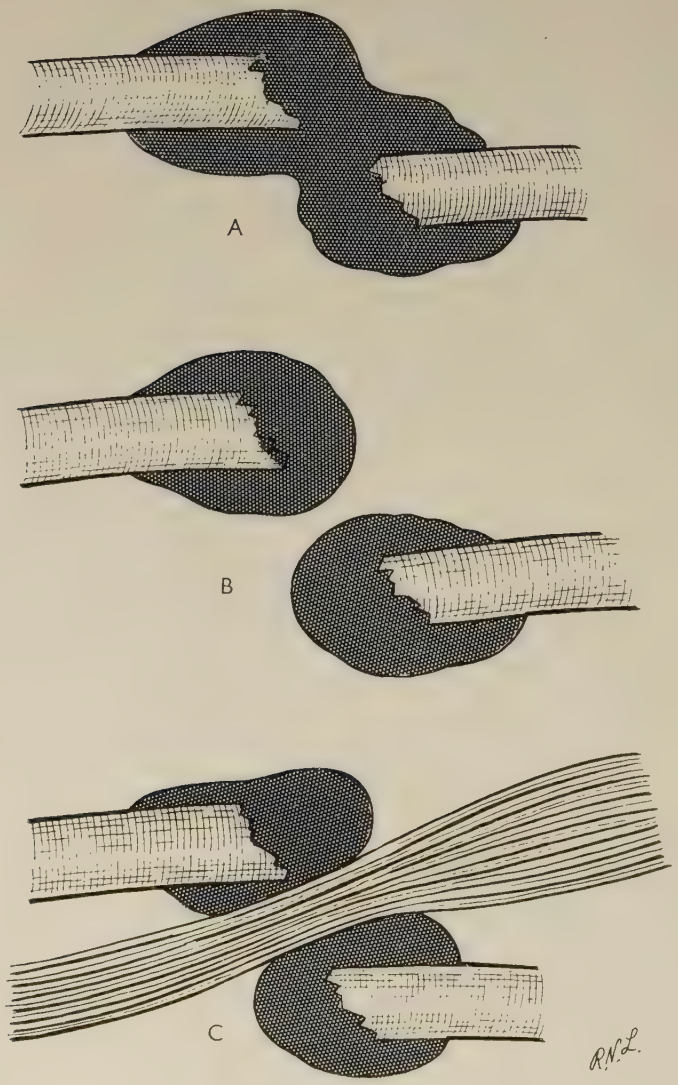

FIG. 9. *Influence of relation of fracture surfaces.* If the fracture surfaces are reasonably close together they are readily enveloped by a single haematoma (A). If on the other hand they are too far apart (B) or if other tissue becomes interposed (C) the haematomata cannot merge and healing will not take place.

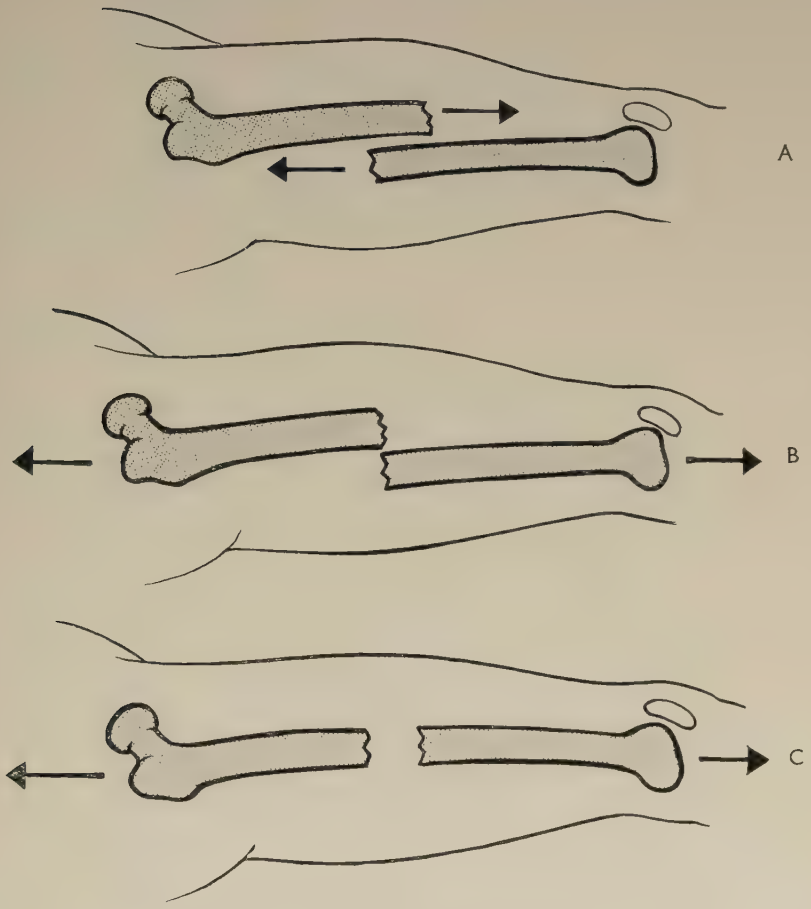

FIG. 10. *Influence of muscle tone and traction.* When a bone is broken muscle tone tends to produce overlap of the fragments (A). This may be corrected by a traction force in the long axis of the bone (B) but if this force is too great or too long maintained the fragments are pulled apart or distracted (C) and this materially delays healing.

simple decrease of the traction force does not reverse the damage, the avascular fibrous tissue which has been formed persists as a barrier to repair.

The exact opposite of distraction, a constant impacting force acting on the fracture surfaces may also have an important effect on repair. It is universally agreed that an intimate, accurate and stable contact between the fracture surfaces is a vital factor in the production of rapid bony union. It has been

further suggested that if these surfaces are actually forced together, that is if the fracture is subjected to a constant compressing force, union is thereby speeded up. There is not, however, universal agreement on the exact effect of compression, and it may be that there is a limit beyond which such a force may be actively harmful to the healing of a fracture.

The relationship between the bone ends also has a bearing on the degree of consolidation of the newly formed callus which is necessary before the bone as a whole can safely be subjected to normal stresses and strains. This question is discussed in a later section.

The relationship between the fracture surfaces is under the control of the surgeon, indeed, the 'reduction' of fractures when displacement has occurred is one of the vital stages in their treatment.

3. *Movement between the bone fragments.* One of the main causes of delay or failure of union is movement between the bone fragments. At every stage of healing any such movement damages the newly formed tissue. The initial enveloping haematoma may be disrupted, the capillary loops joining the bone ends may be torn and the fibrous tissue which forms later may be separated and damaged by any forces tending to alter the relationship between the bone fragments. (*Figure* 11). The exact plane in which movement occurs is not material, the process of repair is equally harmed by a lateral shift, and angulation or a rotatory movement. The range of such movement is important in that the damage done varies in proportion to the amount of displacement which occurs. At the same time any movement, however slight, does damage and delays or prevents natural healing.

If movement between the fragments of a broken bone is allowed to continue indefinitely the nature of the healing process is permanently altered. Because of continued damage to newly formed tissues the gap between the fracture surfaces is not bridged. Instead, separate masses of new tissue develop around each fracture surface and these pass through the stages of vascular granulation, fibrous tissue, cartilage and finally organized bone, sealing off the bone ends completely. (*Figure* 11). When the final stage has been reached no further repair can take place: the bone ends are closed and avascular, soft fibrous tissue separates the fragments and permits movement between them and non-union is said to be established. The area of fibrous tissue between the fragments constitutes a false joint, or pseudarthrosis, at which a varying range of unnatural movement can take place.

The prevention of movement at the site of the fracture during the healing stage is under the control of the surgeon and effective immobilization is one of the main features of the management of any fracture.

4. *Infection.* If pathogenic micro-organisms gain access to the region of a

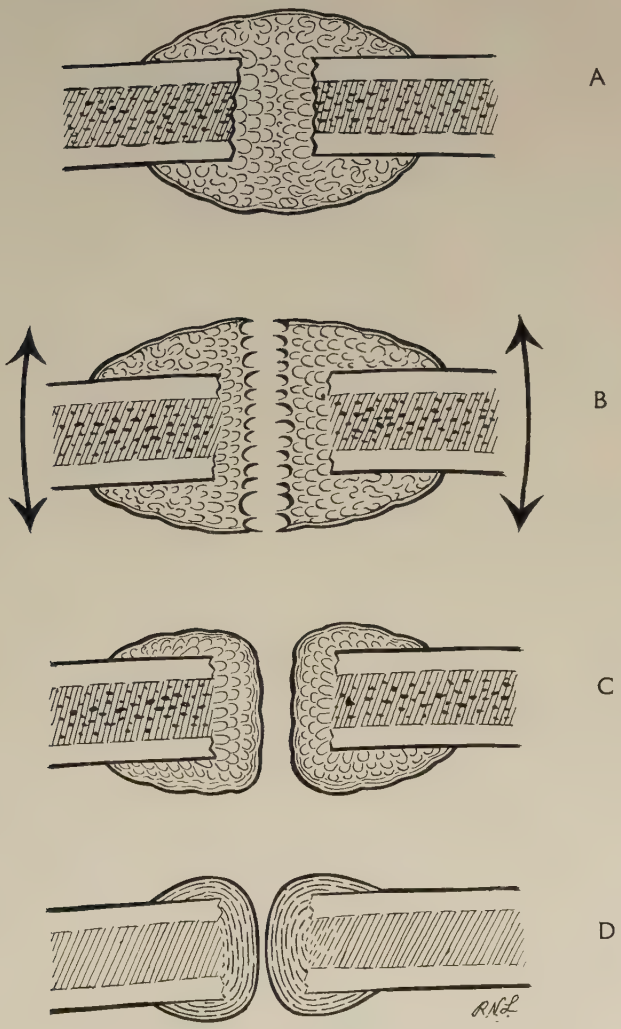

FIG. 11. *Movement and healing.* If, while a fracture is healing, movement is permitted between the fragments (A), the delicate tissues which constitute the early stages of repair are torn (B). Healing then continues independently around each fracture surface without continuity between the bone fragments (C) and ultimately the separate fragments become sealed-off from each other (D). When this stage has been reached no further repair is possible, non-union has become established.

fracture at any stage of healing the inflammatory changes which follow delay and may prevent union.

Micro-organisms may be introduced to the fracture site by two routes. When a fracture is compound, that is when the broken bone is associated with a wound communicating directly with a body surface, direct contamination may occur. Indeed, in most compound fractures some degree of contamination is inevitable and one of the immediate aims of treatment is the prompt elimination of micro-organisms so introduced. Micro-organisms may also infect a simple closed fracture, reaching the fracture site by the blood stream. This may happen when a fracture is associated with an active focus of infection elsewhere in the body.

The presence of pathogenic micro-organisms modifies normal healing in that the changes associated with local inflammation are superimposed on those of repair. The whole affected region becomes hyperaemic, white blood cells migrate to the area, fluid escapes to and distends the tissue spaces and death of individual cells or whole areas of tissue may take place. Death of a sufficiently large amount of body tissue may be followed by local abscess formation.

These inflammatory changes are not confined to the area occupied by the newly formed tissue of repair but involve all the adjacent structures. In particular, the bone ends in the region of the fracture surfaces are infected by micro-organisms and inflammatory changes spread into the bones themselves. These changes have a particularly important influence on the further course of repair. It is universally true that any marked increase in the blood flow through living bone produces a decalcification of the area of bone affected and the inflammatory hyperaemia occurring in the infected area is followed by just such a decalcification. If the infection progresses unchecked, the decalcified area of bone may actually die and the dead bone is either absorbed or persists as a sequestrum. Inflammatory destruction of bone in this way may convert a fracture into a large bony defect and even if the infection later resolves the gap so produced may be too great to be bridged by normal repair.

The effects of infection of a fracture by micro-organisms are, therefore, always harmful and may be catastrophic and the prevention or control of such infections is an important aspect of the treatment of fractures.

5. *Other factors.* Normally not many other factors affect the healing of a fracture but there are certain general conditions which may be of significance.

The age of the patient has already been discussed. The growing bones of children unite more readily than adult bones, and the more rapidly a bone is

growing, that is the younger the patient, the more quickly does it unite after it has been broken. The effects of age become less marked as the patient gets older and by the late teens the bones have become more or less adult in their behaviour.

In the normal healthy person the body contains an ample supply of all the substances necessary for the union of fractures and even in the case of multiple injuries when many bones are broken repair takes place quite normally. In the case of deficiency diseases or after a period of prolonged starvation, however, healing of bone, like other healing, may be delayed. For example, in the inmates of concentration camps general decalcification of the skeleton often occurred and these brittle bones fractured easily and subsequently united slowly and imperfectly. No drug or other substance is known which can, when administered to a normal person, speed up the healing of a fracture. Obviously, such a compound would be of the greatest value and hundreds of substances have been tried to this end without success.

While nothing is known which will help to make a fracture unite the reverse is by no means the case and quite a number of substances which can be introduced into the body may delay or prevent union. For example, the anticoagulants, which might be used for certain complications occurring after injury, may delay the early stages of healing. All the cortisone preparations have a disastrous effect on normal fracture healing at all stages and in view of the wide-spread and mainly undeserved reputation enjoyed by this group of drugs and the present vogue for their empirical and wholly irrational use in all sorts of conditions this point is worth remembering.

In a healthy person, therefore, no substance need be administered to promote bony union, but certain drugs must be avoided. If, however, there is any body deficiency due to disease or malnutrition, this must be made good as part of the treatment of a fracture.

OTHER CHANGES ASSOCIATED WITH FRACTURES

Any force of sufficient violence to break a bone must traumatize soft tissue as well. This associated damage may have an important relation to the progress and end results of any fracture. The tissues most affected are usually those in the immediate vicinity of the fracture itself where structures attached to or in contact with the bone are directly involved. When fractures are produced by indirect or transmitted forces, however, associated injury may occur at the point where such forces are applied and may be quite distant from the fracture site. In certain types of injury, notably of the crushing variety, damage to soft tissues far outweighs the bony injury in importance.

The changes in the different types of body tissues in association with fractures may be considered in more detail.

Oedema. Following a fracture, generalized swelling of all the tissues in the vicinity takes place and is caused by fluid leaking from the capillary bed into the tissue spaces. It starts within a few moments of injury and may become very widespread. For example, injury to one of the bones of the arm or leg may be associated with a generalized gross swelling of the whole limb. Normally such swelling reaches its maximum within five or six days of injury and then gradually subsides but the extent and duration of the oedema vary widely not only with the nature and severity of the associated fracture but also with the individual patient. Both the degree and duration of the swelling associated with a fracture can very largely be controlled by treatment.

Skin changes. In those fractures produced by direct violence the skin is usually directly injured and may be contused, abraded, split or even cut depending on the degree and nature of the force involved. Even if direct injury does not occur, however, the skin is often affected by the oedema which may follow a fracture. When swelling is considerable widespread vesiculation of the skin may occur, and the presence of these so-called 'fracture blisters' may embarrass treatment.

Subcutaneous tissue. Bruising, sometimes associated with widespread extravasation of blood, particularly in the elderly, and swelling are the main changes occurring in subcutaneous tissue following a fracture.

Muscles. Practically every fracture involves the adjacent muscles in some degree. At best they are contused so that local bleeding and oedema take place around their fibres. Usually, when one of the long bones is broken muscle fibres are actually torn or lacerated, either at their points of attachment if they are involved in the fracture or as the result of penetration by the bone ends. Contact between the bone ends may actually be prevented if muscle becomes interposed between them or if one end becomes embedded in muscle tissue.

Two other conditions may affect the muscles in association with fractures.

New bone formation in the region of a fracture, the so-called 'myositis ossificans', may follow any injury involving both muscles and bone. Such posttraumatic ossification does not occur primarily in the muscle itself but takes place in any haematoma associated with broken bone or avulsed periosteum or bone fragments. Such periosteum or fragments act as a source of osteoblasts which invade the organizing blood clot. The subsequent changes are, in fact, exactly similar to those occurring in normal bone healing but without a complete fracture the new bone so formed serves no useful function. In

these circumstances the distribution of the haematoma determines the site of subsequent deposition of bone. If this haematoma is small and localized or if it is eliminated by surgical repair of the damage subsequent ossification is unlikely to do any harm. If, however, the haematoma is widespread and, particularly, if it is distributed and dispersed around adjacent muscles and other tissues by injudicious treatment extensive scattered new bone formation will take place and the function of the muscles involved is permanently affected.

A second and most significant and extensive series of changes may take place in muscle tissue in association with bone injury. If the arterial supply to a group or groups of muscles is cut off, either by vascular spasm or by direct involvement of a main artery in a fracture, death of the muscles so affected takes place quite rapidly and the dead muscle fibres are subsequently replaced by fibrous tissue. These changes are associated with complete loss of the normal elasticity and contractile function and with marked shortening of the affected muscle groups. There is subsequent gross deformity and loss of function and this condition, a Volkmann's Ischaemic Contracture, is one of the most serious complications which can occur.

Tendons. Direct injury to the muscle tendons is rare in association with a fracture but any generalized swelling is associated with an effusion of synovial fluid within the tendon sheaths. If the tendons are allowed to remain immobile while this effusion resolves they become adherent to the walls of their sheaths and part or all of their normal range of movement is lost.

Blood vessels. Fractures may be associated with various arterial complications. The most common of these is arterial spasm, occurring mainly in association with fracture of the long bones in the limbs. Such spasm usually affects a segment of a main artery at or immediately distal to the fracture level. Spasm has, however, been reported proximal to the fracture line and often quite long segments of a vessel are affected. Arterial spasm is probably almost always the result of direct injury to the affected artery, often in the form of a traction strain or a kinking following displacement at the fracture site. An artery may occasionally be directly involved when a bone is broken. If the vessel is severely contused subsequent intra-arterial clotting may occur, blocking the blood flow. The artery wall may be pierced by bone splinters and an immediate leak or a subsequent aneurism may be produced in this way. An artery may also be trapped in a displaced fracture and so compressed or kinked as to be completely occluded. On rare occasions complete rupture of an artery may occur.

However produced, the interruption of the blood flow through a main artery is a most serious complication. Unless an adequate collateral circula-

tion exists the consequences are always grave and complete gangrene of an entire limb may occur.

The veins may also be involved in changes associated with fractures, contusion with subsequent thrombosis or, later, thrombosis associated with stasis or immobility being the most common complications. The veins may also be torn or cut and occlusion of the main veins in a limb, while not as serious as arterial obstruction, is followed by swelling and water-logging of the whole limb.

Embolism. Immediate or late intra-arterial or venous thrombosis may be followed by embolism. Embolism due to a blood clot is not an excessively rare complication of fractures. Less commonly, when one of the larger long bones is broken fat embolism may take place. This occurs when fat globules from the marrow cavity gain access to the circulation through ruptured vessels.

Nerves. The peripheral nerves may be involved in the injuries associated with a fracture.

Disturbances of the blood supply, already discussed, may deprive a nerve trunk of blood with subsequent loss of nerve function.

Nerve trunks may also be involved directly in a fracture in the same way as the vessels, they may be stretched, compressed or lacerated by the bone ends. The result of such injuries is the interruption of the conductivity of some or all of the nerve fibres with consequent loss of nerve function. Such an interruption may be temporary, as follows a contusion, when the nerve fibres recover within a matter of days without undergoing any degenerative changes, or permanent, when the fibres distal to the site of injury die and function can only be restored by the growth of new nerve fibres from the severed proximal end. Two types of injury are associated with this form of complete loss of function, anatomical section when the nerve trunk is severed in its entirety, and physiological section when only the nerve fibres are affected, their supporting sheaths remaining intact. After anatomical section recovery can only take place if the severed nerve ends are brought into close and stable contact; and even so regeneration after surgical repair is slower and more imperfect than regeneration after physiological section.

Both anatomical and physiological section of a nerve trunk may, of course, be partial, some fibres only being affected.

Division of a nerve trunk, either temporary or permanent, is followed by complete loss of both sensory and motor function with subsequent wasting of the muscles concerned and numbness and trophic changes in the skin area supplied.

The involvement of a nerve in a fracture is not necessarily immediate.

Subsequent callus formation may impinge on adjacent nerve trunks or bony displacement be followed by friction between a nerve and underlying bone. In both these conditions conductivity of the involved trunk may be interrupted but in these circumstances the changes are gradual and the subsequent loss of function and the onset of symptoms is an insidious and slowly progressive phenomenon.

Joints. When a bone is broken the plane of the fracture may cross an articular surface. The joint concerned is then directly involved in the bony injury and, if displacement occurs, may be mechanically disorganized. This type of injury is discussed in the section dealing with the joints.

Apart from direct injury adjacent joints are always to some extent affected indirectly when a bone is fractured. Violence sufficient to break a bone must be transmitted to these joints and produce changes. Within the joint a traumatic synovitis and an effusion of synovial fluid is usual, and if the injury is severe the articular cartilage may actually be contused. Periarticular oedema and reflex inhibition of the muscles moving the joint also often occur after injury. Later as coagulation of the exudate occurs, the periarticular tissues may become adherent to each other and there may be some fibrous tissue formation, with consequent limitation of joint movement. Wasting of the inhibited muscles is usual.

Generalized changes following a fracture. While it is simple to discuss the individual changes in separate tissues following bony injury the overall picture, particularly in its late stages, is even more important. Indeed, after a fracture the ultimate result from the functional point of view is determined as much by the state of the soft tissues as by the way in which the bony damage has been repaired. These generalized changes are discussed more fully later.

SPECIAL CHANGES ASSOCIATED WITH FRACTURES

There are three other special phenomena which may occur after a bone is broken. Two of these affect the bone only and the third is a more generalized change affecting both bone and soft tissue.

Bone absorption at the fracture surfaces. Commonly a bone is broken but not displaced, that is the fracture surfaces remain in close, accurate and intimate contact. On X-ray examination immediately after injury such fractures appear as a crack, a line extending across the normal bony trabeculae. If such fractures are not immobilized or are inadequately immobilized within a few days absorption in the region of the fracture line may take place so that the defect becomes much greater and more obvious on X-ray examination.

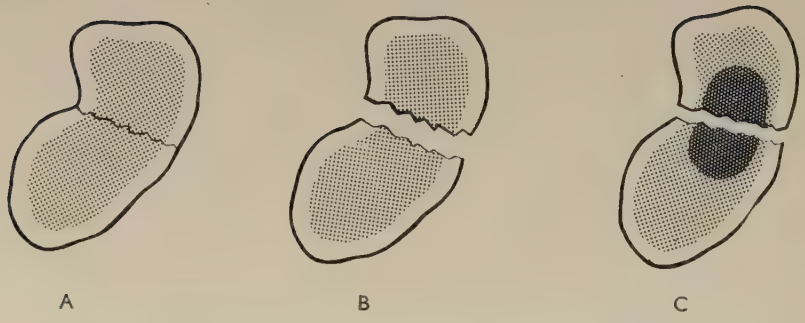

FIG. 12. *Bone absorption at the fracture site.* A recent fracture of the carpal scaphoid appears as a linear crack (A) but if untreated or inadequately treated absorption occurs at the fracture surfaces and a defect appears between the bone fragments (B). In certain circumstances bone absorption continues and a cavity may appear in the centre of the bone (C).

This absorption is not always confined only to the region of the fracture; for example, in an untreated fracture of the carpal scaphoid a cyst-like defect may occur in the centre of the bone. (*Figure* 12).

The usual explanation of this phenomenon is that the friction between the unsplinted fracture surfaces produces local irritation and hyperaemia and that this hyperaemia is followed by decalcification. It is doubtful if the reason for bone absorption of this kind in the region of a fracture line is quite as simple as this. Infection by micro-organisms, with consequent hyperaemia, is followed by a similar type of bone absorption but infection in simple fractures is rare. Inadequate immobilization may be a contributory cause but quite often completely untreated fractures show no such changes. Moreover some absorption is occasionally seen in fractures, even in children, when immobilization has been completely adequate.

It may be that the value claimed by some surgeons for a compressing strain between the fracture surfaces as an aid to bony union depends on the fact that such a force would at once obliterate any defect produced by bone absorption in the region of the fracture line.

Bone loss around a fracture is always associated with delay in union but provided adequate immobilization is continued for a sufficiently long period normal consolidation of the fracture will ultimately take place.

Avascular bone necrosis. Broadly speaking all bones have a rather lavish blood supply, and the free anastomosis around and within them makes it unlikely that any individual part will be entirely deprived of its blood supply when the bone is broken in the usual way. In certain bones, however, arterial

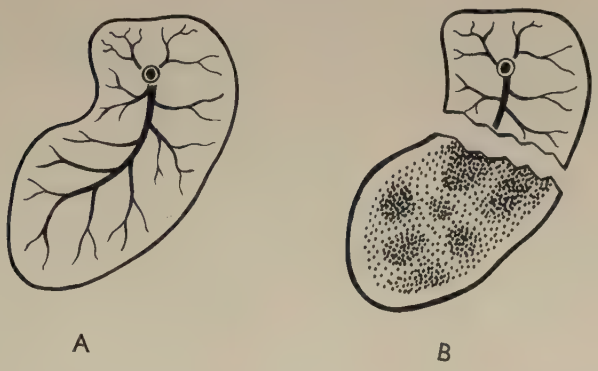

FIG. 13. *Avascular bone necrosis.* If part of a bone is cut off from its blood supply by a fracture (A) the avascular fragment undergoes a cycle of changes to which the term avascular bone necrosis is applied (B).

distribution is such that a fracture may cut off quite a large fragment from any blood supply (*Figure* 13). When this happens the avascular bone undergoes a cycle of changes to which the term 'avascular bone necrosis' is applied.

Deprived of blood the affected bone quickly dies. Its 'hard' framework remains but contains no living cells. At first the architecture of the bone is unaffected but if subjected to continual stresses and strains the 'dead' fragment may become compressed or distorted. The radiological changes at this stage are of some importance. The calcium content and therefore the density of the bloodless fragment remains unaltered but the surrounding bone often loses some density because of post-traumatic hyperaemia. In these circumstances the avascular fragment appears on radiological examination to be relatively sclerotic in relation to the surviving bone. Unless and until it has become distorted by pressure the bony architecture of this fragment remains normal.

In favourable circumstances the 'dead' fragment is slowly invaded by capillary growth from adjacent vascular bone and a process of slow regeneration takes place. The dead bone acts as a scaffolding by and from which repair takes place and is gradually converted into living bone. This process, which is exactly the same as that which occurs when an autogenous or homologous bone graft is inserted, is slow but ultimately the avascular fragment is completely replaced. The new bone formed in this way is normal in every respect but it assumes the shape of its 'scaffolding', that is distortions due to pressure are not corrected.

While regeneration of bone is complete this is not true of articular cartilage attached to the affected fragment. Like bone the articular cartilage dies when it is deprived of its blood supply but unlike bone it does not regenerate when this is re-established but is replaced by fibrous tissue.

'*Sudecks atrophy*'. Sound bony union of a fracture in a limb, notably in the forearm, is not invariably associated with recovery of function. Various degrees of disfunction may occur but in its most florid form this condition is characterized by widespread decalcification of bone, stiffness of all the joints of the limb, muscle spasm, muscle wasting, oedema and trophic changes in the skin. To this syndrome the name of Sudecks post-traumatic acute bone atrophy has been applied.

The aetiology of this complication is unknown. Some authorities even deny its existence and assert that all these changes are the results of simple functional disuse and can be prevented by adequate treatment. It is difficult to accept this, however, as from time to time, particularly in elderly patients, this syndrome may follow a fracture which has been adequately treated in every respect.

2

THE PRINCIPLES OF TREATMENT OF FRACTURES

THE PRIMARY purpose of the treatment of any fracture is the ultimate restoration of the function of the injured part to normal, or to as nearly normal as possible. This might seem a simple and obvious enough fact but it is surprising how often it is lost sight of by those engaged in the management of these injuries.

To this primary aim there must be added various secondary objectives. Treatment should relieve the patient's pain or discomfort, it should also minimize the immediate disability as far as possible, and finally it should be designed to expedite final recovery so that the total period of disability is reduced. These latter are, however, essentially secondary considerations and measures to these ends are permissible only when they do not interfere with or jeopardize the success of the primary aim of treatment.

Essentially, the management of fractures consists of the establishment of the best possible conditions for natural repair of the broken bone and for the healing or resolution of the associated changes in the soft tissues. At the same time measures must be taken to prevent the development of any other harmful changes during the healing period.

FACTORS WHICH DETERMINE SUBSEQUENT FUNCTION

Obviously the logical treatment of fractures must be based on a knowledge of the factors which will ultimately determine the function of the injured part, and these will now be discussed.

1. *Bony union.* Unless and until the fragments of the broken bone become united by solid bone sufficiently strong to stand up to all the forces to which it may normally be subjected function cannot be restored to normal.

If union is not solid function is affected in three ways: the unstable fracture site is a source of pain when subjected to stresses; there is inhibition and spasm of all the muscle groups acting on the bone fragments; and the stability normally provided by the intact bone is lost.

There are, of course, many degrees of failure of bone union with consequent variation in the intensity of the symptoms produced. Repair may have

failed altogether and a pseudarthrosis may exist allowing a relatively free movement between the bony fragments. Healing may be incomplete, the fragments being joined by fibrous tissue which stretches if subjected to stresses and allows limited movement at the fracture site. The callus formed may be soft, so that it bends under strain with alteration in the relation of the bony fragments. Not only is function affected by any of these circumstances, but if an incompletely consolidated fracture is subjected to continuous or repeated strains, retrograde changes occur, the healing process is reversed and the fracture becomes increasingly unstable with a consequent further deterioration in function.

The degree of union of any fracture is tested in two ways, by clinical and by radiological examination. On clinical examination the bone is subjected to manual strains and if these produce local pain at the fracture site or movement between the fragments union is not complete. On radiological examination the amount and density of new bone formation can be assessed from the radiographs. Newly formed callus takes some time to become as dense as normal bone and quite often a fracture may be clinically sound some time before it appears completely consolidated on X-ray examination. In these circumstances the clinical condition may safely be accepted as evidence of union, provided the patient is kept under close observation for the possible development of retrogressive changes.

2. *The relationship between the bone fragments.* The bones are the framework of the body, and any distortion of the framework is associated with deformity and disturbance of function. Function cannot, therefore, be restored fully unless a fracture unites with the bone fragments in normal relationship to each other. If displacement is allowed to persist disfunction is inevitable and its degree is usually proportional to the amount of displacement. The effects of malposition of the bony fragments are particularly important in the limbs where it is associated with disfunction of the joints above and below the fracture site.

As far as long bones are concerned there are three main types of displacement although these usually occur in combinations.

Fragments may be in normal alignment but may overlap. When a fracture unites in this way the bone is shorter than normal, with a consequent shortening of the limb as a whole. In the arm, such shortening is of little or no consequence unless it occurs to a very unusual degree; but in the legs any significant loss of length is followed by a limp, pelvic tilting, scoliosis and other secondary changes associated with shortening of one leg. The greater the shortening the more marked are these changes. (*Figure* 1).

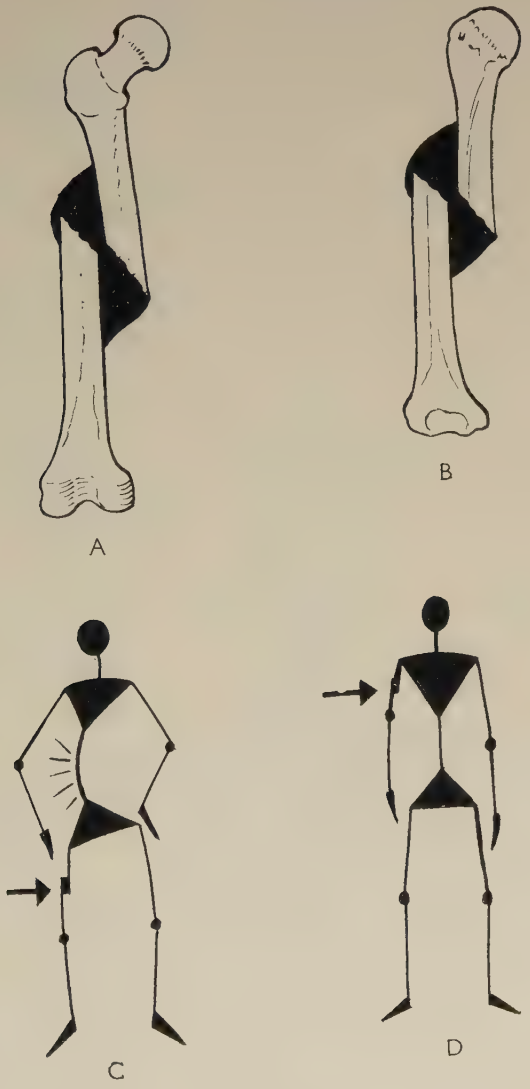

FIG. 1. Fractures of the shafts of long bones may unite with overlap and consequent shortening of the bone as a whole (A and B). In the leg such shortening is associated with a postural deformity affecting the whole body and with disfunction of various kinds (C). In the arm, on the other hand, only the injured limb is affected and any loss of function is trivial (D).

The second type of displacement is an angulation in the long axis of the fragments. Such an angulation may disturb the muscles crossing the fracture by altering their direction of pull but its main effect is on the joints above and below. The plane of these joints, in relation to the long axis of the limb, is abnormal with an immediate disturbance of their movement. Subsequently, particularly in the leg, the distorted joints are subjected to forces which fall on them in an abnormal direction, and these forces damage the articular cartilage and produce progressive degenerative changes. (*Figure* 2).

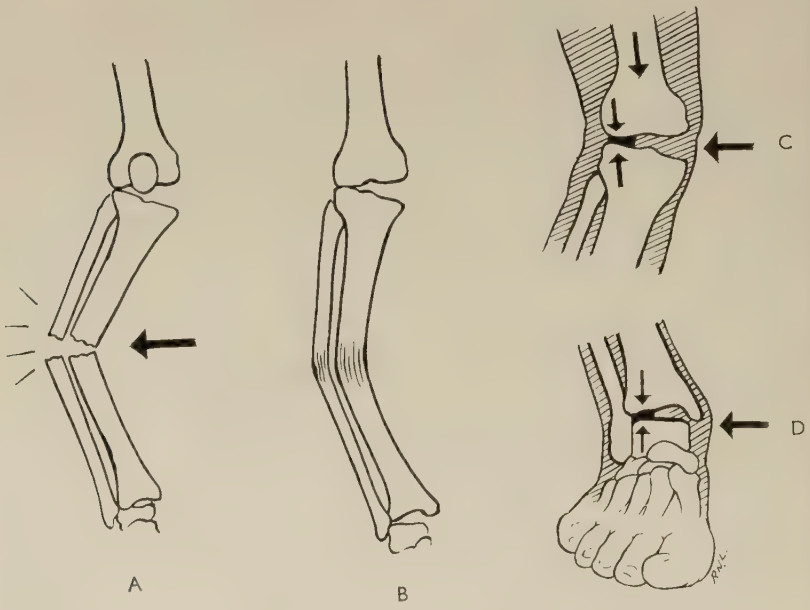

FIG. 2. If a fracture of a long bone is allowed to unite with an angulatory deformation (A and B) the plane and function of the joints above and below are disturbed (C and D).

The third, and most insidious, type of displacement is in a rotatory plane. This displacement may be overlooked completely at the time of injury because it is not immediately obvious on clinical observation and may not show at all on an X-ray. Like angulation, rotation may disturb the action of the muscles crossing the fracture, but its main effect is on the joints above and below. The planes of movement of these joints no longer correspond and function is disturbed. (*Figure* 3).

In small bones the relationship between the fragments is of somewhat

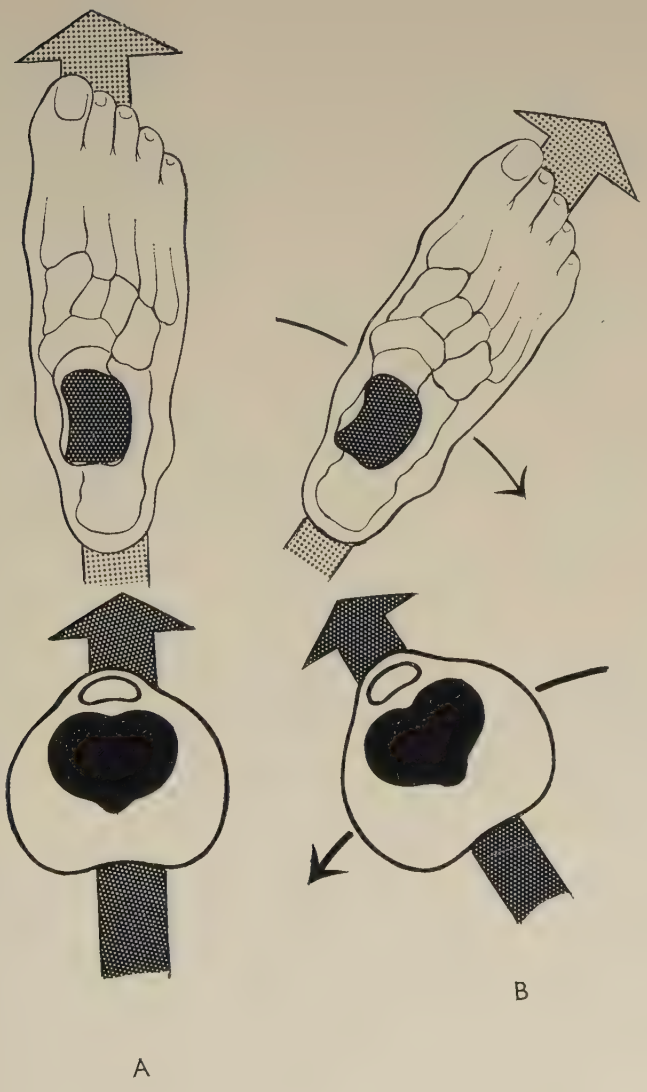

FIG. 3. Normally the joints in a limb move in the same plane (A). If a fracture of the long bones is allowed to unite with a rotatory deformity these planes no longer correspond (B) and function is disturbed.

less importance, and subsequent disfunction is usually only produced when the fracture line involves an articular surface which is made irregular by displacement.

The local relationship between the fracture surfaces is of less importance to ultimate function than that of the fragments as a whole. Ideally, of course, these surfaces should be in close and accurate contact but this is mainly because rapid and solid union is thereby promoted. It is, in fact, possible for the fracture surfaces to be in partial contact or even not in contact at all without subsequent disfunction provided union takes place. (*Figure* 4). Lack of

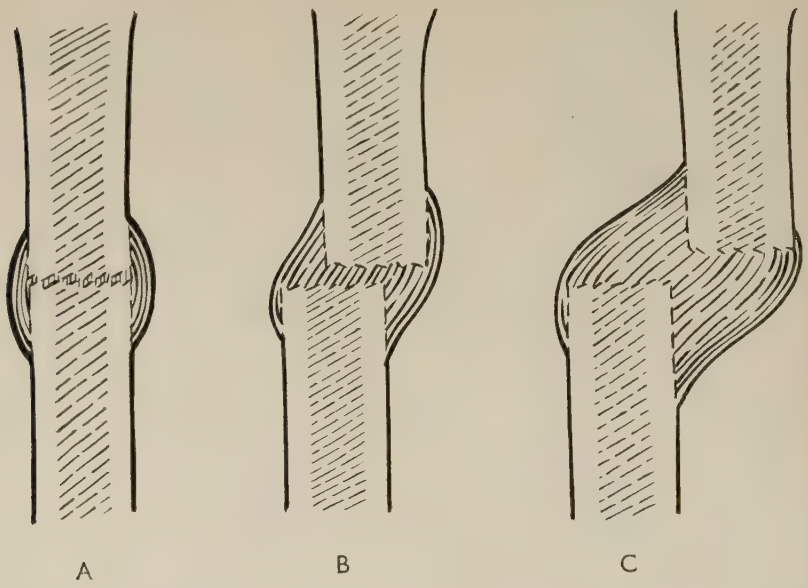

FIG. 4. The local relationship between the fracture surfaces does not determine whether or not union takes place. Fractures unite best when the bone ends are in accurate and close contact (A) but union can take place when they are in partial contact (B) or even not in contact at all (C).

accurate apposition has some other effects, however. The extensive repair made necessary may be associated with local adherence of muscle and other soft tissues with consequent disturbance of function. When healing has taken place and the fracture is exposed to strains these fall entirely on newly formed tissue and unless this is well consolidated retrogressive changes may occur. (*Figure* 5). Finally lack of contact may be a source of subsequent trouble

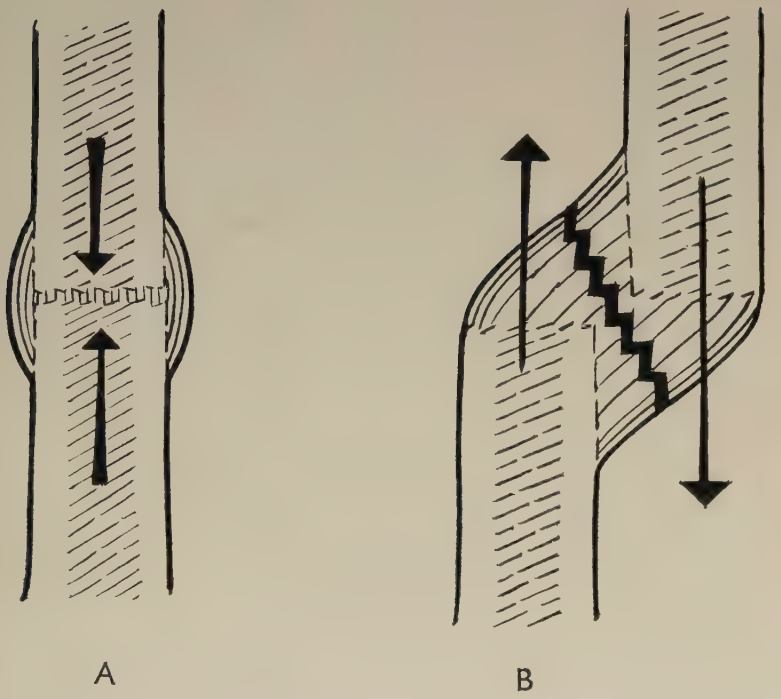

FIG. 5. If a fracture has healed with the fracture surfaces in close contact when it is exposed to strains these are transmitted through the bone itself (A) and do no harm. If the bone ends are not in contact but joined by callus such strains fall directly on the newly-formed tissue and unless this is well consolidated retrogressive changes may take place (B).

because, unless remoulding is very extensive, the local deformity may, especially in the case of a subcutaneous bone, produce a vulnerable and easily injured swelling.

3. *The state of other tissues.* Solid union of a fracture with the bony fragments in normal relationship is not of itself a guarantee of good function. The adjacent soft tissues and, in the limbs, the associated joints are affected both by the initial injury and by the period of immobilization and disuse or partial disuse necessary to permit healing of the fracture. Normal function depends equally on restoration to normal both of the bone and of all the associated tissues and structures.

The changes in other tissues have already been discussed. Muscle wasting and weakness occur as the result of disuse and injury and if the muscles ad-

here to the fracture site their range of movement is decreased. Tendons may adhere to their sheaths and to surrounding structures, with consequent loss of movement. The joints above and below the fracture may become stiff as the result of fibrous tissue formation in the capsule and in the peri-articular structures. If vascular changes have occurred these may be followed by the phenomena associated with circulatory deficiency. Peripheral nerve injury is followed by partial or complete paralysis of muscle groups in the case of a motor nerve, and by anaesthesia and trophic changes of the skin when a sensory nerve is involved. Any gross damage to skin or soft tissue may cause extensive scarring with consequent contraction and stiffness.

The presence of some or all of these changes may be associated with gross disfunction and their prevention or reversal is one of the main purposes of treatment.

THE PRINCIPLES OF TREATMENT OF FRACTURES

There are three basic principles on which the treatment of all fractures depends. While the details of management may vary greatly, these principles are absolute and must never be neglected or ignored if the results of treatment are to be satisfactory.

1. *Reduction of the fracture.* If, when a bone is broken, there is displacement at the fracture site the first step in treatment is to restore the normal relationship between the bone fragments, that is to reduce the fracture. This is done to prevent subsequent disfunction due to persistent deformity and to establish conditions favourable to bony healing.

Ideally, of course, the displacement should be reduced completely so that the fracture surfaces are brought into close and accurate contact with the bone as a whole in its normal shape. One must recognize, however, that many different types of displacement exist and these are not of equal importance either to union or subsequent function. Angulation or a rotatory displacement between the main fragments is always associated with subsequent disfunction and must not be allowed to persist. The exact relationship between the fracture surfaces is less important. Partial contact or even no immediate contact may be compatable with normal function. Some degree of overlap may also be permissible; but separation in the longitudinal plane of the bone, that is distraction, interferes with healing and must not be accepted.

An adequate reduction may, therefore, be defined as a relationship between the fragments which will not produce disfunction or interfere unduly with the normal process of healing.

There are three ways in which fractures may be reduced.

(A) *Manipulation.* By far the simplest and safest way to reduce a fracture is by manual manipulation of the fragments. In skilled hands the vast majority of fractures can be reduced in this way without danger of further damage to soft tissue or bone. The patient is anaesthetised, using either general or, in special circumstances, local or block anaesthesia, and the surgeon and his assistants with their hands move the bony fragments into a normal relationship. Various manoeuvres are necessary to do this and different fractures present different problems.

Reduction should be carried out as soon as possible after injury. If gross oedema is present it is more difficult to feel and control the fragments, but it is quite unjustifiable to postpone reduction, as is so often done, 'until the swelling goes down'.

Accurate manual reduction of fractures can only be ensured by the intelligent use of X-rays. Radiographs taken after injury show the displacement requiring correction, while those taken after manipulation show whether this has been successful or must be repeated. In some circumstances, manipulation may actually be controlled by radiographs taken during the process; but with increasing experience most surgeons find this unnecessary.

A successful manipulation and an accurate reduction does not end this particular problem, however. Very few fractures which have been displaced are stable on reduction, and until healing has become fairly well advanced re-displacement may occur for a number of reasons. The prevention of re-displacement is fully discussed later but it is essential that, if it should occur, it be promptly detected and the fracture re-reduced. For this reason, after reduction the fracture is X-rayed at regular intervals until the danger of re-displacement has passed.

Manipulation under anaesthesia may sometimes be employed in the case of fractures which show no displacement. In these circumstances the patient is anaesthetised and the fracture is gently moved to determine whether or not it is stable, that is to say whether displacement occurs easily or not. The degree of stability may influence subsequent treatment, and it is for this reason that such examinations are undertaken in certain fractures.

While manual reduction is usually employed, from time to time efforts are made to introduce more mechanistic methods. Usually the fragments above and below the fracture are transfixed by a varying number of pins which are then fixed to a mechanically controlled frame and moved in relation to each other under radiological control. These engines, symptomatic of this machine age, enjoy a brief vogue and are then retired to join their predecessors and be joined, in due course, by their successors. When dealing with injured tissues, no mechanical device can compete with the human hand.

(B) *Continuous traction.* A long bone which is deeply buried in muscle may be impossible to reduce by manipulation. In certain cases the most desirable alternative is continuous traction. The displacement to be corrected in this type of fracture is usually overlap produced by muscle tone, often associated with an angulation or rotation between the fragments. Traction is applied in the long axis of the bone and gradually increased over a period of two or three days, the position of the fracture being checked by a series of X-rays. As the overlap is corrected by increasing traction the angulation also tends to be corrected, but usually lateral pressure applied by various splints is also necessary. Reduction should be complete within a week, and the distracting force is then stabilized so that it is just sufficient to maintain the position. Traction is applied to the bone in one of two ways. The least efficient is skin traction, the whole limb being pulled on by means of strips of adhesive strapping applied to the skin. Much more effective is direct skeletal traction using a transfixing pin inserted through bone.

Formerly, continuous traction was fairly widely used in the treatment of fractures but today its use is almost entirely confined to fractures of the shaft of the femur.

(C) *Open reduction.* It is not always possible to effect reduction by any form of manipulation. This is usually because soft tissue has become interposed between the fracture surfaces, or the end of the fragment has become buried in a muscle, but sometimes a fragment is too small to grasp or control. A double or multiple fracture of a long bone may also be uncontrollable by manipulation.

When this happens, open reduction of the fracture becomes necessary. The site of injury is exposed by operation, and, under direct vision, the bone ends are cleared and restored to their normal relationship. When the bones are exposed in this way it is, of course, possible to achieve complete and accurate reduction with certainty. Alternatively, if there is a small fragment which is not essential to function it is sometimes desirable to remove it completely and suture the attached soft tissues to the main fragment of the bone.

Open reduction is also practised in one other circumstance. For reasons discussed in a later section, internal fixation is necessary in certain fractures and this may involve exposure of the fracture site. If this has to be done the exposed fracture is, of course, reduced under direct vision at operation.

Although open reduction is probably the most accurate of all methods of correcting bony displacement, it is associated with various hazards which are discussed later. For this reason it should never be undertaken when a reduction can be achieved successfully by simpler means.

2. *Immobilization of fractures.* The first step in the treatment of a fracture

is the reduction of any displacement which may exist. This having been done, the fracture must be immobilized until healing has taken place. This, the second cardinal principle in the treatment of fractures, means that the broken bone must be fixed in such a way that movement between the fracture surfaces cannot take place and the healing tissue is protected from all mechanical stresses.

Immobilization has two primary purposes. Re-displacement between the fragments and subsequent disability on this account is prevented. Equally, as has been pointed out, any movement or tendency to movement at the fracture surfaces prevents or delays healing and adequate fixation, therefore, promotes and accelerates bony union. Immobilization also relieves pain and, if suitably devised, permits functional activity while the fracture is healing.

All fractures must be immobilized but not all require the same amount of protection. Broadly speaking, they fall into two classes, stable and unstable. A stable fracture is not easily displaced, considerable force being necessary to produce movement at the fracture surfaces. An unstable fracture, on the other hand, displaces very easily. Any body movement, the contraction of muscles or even normal muscle tone results in movement between the fragments. It should be noted that this grouping of fractures into stable and unstable does not correspond with the other usual grouping into undisplaced and displaced. It is true that practically all displaced fractures are unstable after reduction but undisplaced fractures are not all stable by any means. The importance in some circumstances of the examination under an anaesthetic of an undisplaced fracture to determine its stability has already been discussed. Further, there are many different degrees of instability. Usually transverse fractures, either undisplaced or after reduction, are relatively stable, while oblique or comminuted fractures are very difficult to control. A particularly insidious type of instability is that in which rotatory movements tend to take place between the fragments.

All these factors must be taken into account by those engaged in the management of bony injuries and the type of fracture often determines the measures employed to effect its fixation.

There are three basic ways in which a fracture may be immobilized.

(A) *External fixation.* The external fixation of a fracture, that is the application of a rigid splint to the body or to a limb when a bone is broken, is probably one of the earliest forms of medical treatment practised by man. The principle is simple enough, a rigid structure is applied as closely as possible and fixed firmly in position to provide the stability normally associated with an intact bone. The effectiveness of this method depends on several factors, and it has certain obvious dangers and disadvantages. To work efficiently, the

rigid structure must be closely fitting and must be fixed firmly in position. It must extend well above and below the site of the fracture, the usual principle being that the joints above and below must be included in the area immobilized. Difficulties inherent in the method are chafing of the skin, pressure between the splint and underlying bony points and the achievement of firm fixation without undue constriction of the limb.

Formerly external fixation was achieved by the use of splints. In their crudest form these were improvised out of wood or metal and were held in position by straps and buckles or bandages. The discovery of new and more effective splints afforded almost unlimited scope for ingenuity, however, and countless different types were employed in the treatment of fractures at various times and in different countries.

The limitations of these splints became obvious in the early years of this century when radiology came into general use. The use of X-rays made it possible to reduce fractures with accuracy but re-displacement occurred far too frequently. The demand for a more efficient technique for external fixation was met by the re-introduction of a very old method, the use of some substance which could be applied in a soft or malleable state and which would set hard quickly. Bandages impregnated with Gypsum or Plaster-of-Paris, when soaked in water and applied to the body, quickly set hard. When set, the plaster remains hard and wears well for some weeks. It is not too heavy, rarely produces skin reactions and its chief disadvantage is that it becomes soft when exposed to moisture.

Plaster-of-Paris is now almost universally employed for the external fixation of fractures. The technique of its use varies considerably. Formerly, under the influence of the Vienna school, the injured limb was completely encased in plaster, the mis-called 'plaster cast', applied directly to the skin. These unpadded plasters were not without dangers however, and it is now unusual to apply plaster directly to the skin without some form of intermediate padding.

The technical details of the use of plaster are discussed in a later section.

(B) *Traction.* The use of continuous traction either directly to the skeleton or through the skin in the reduction of certain fractures, notably those of the femoral shaft, has already been discussed. When reduction has been achieved by this means, the traction is not usually discarded but is continued as a means of external fixation. When any shortening present has been corrected the traction force is decreased until equilibrium is reached, and is then maintained at this level until the fracture has united. Careful radiological control of the traction force until stability has been reached is essential, the dangers of separation of the bone ends have already been referred to.

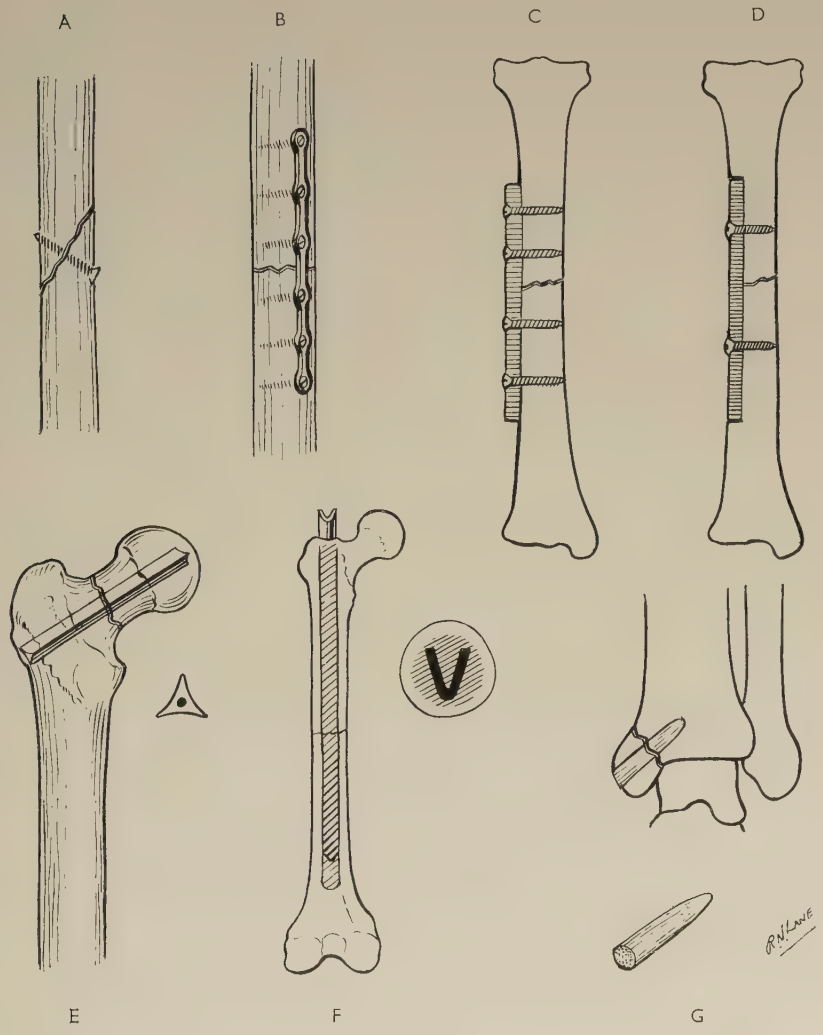

FIG. 6. When a fracture has been exposed and reduced the fragments may be fixed together in many different ways. Some of the most commonly used methods are a single screw (A), a plate and screws (B), an onlay bone graft fixed with screws (C), an inlay bone graft (D), a trifin nail (E), a U-shaped intra medullary nail (F), and autogenous peg (G).

Traction alone is not enough to control a fracture and it is used in conjunction with some form of external fixation, for example, the Thomas splint.

This method of immobilization is not very widely applicable and requires constant supervision and skilled adjustment. In experienced hands, however, it is the most effective way to control certain fractures.

(C) *Internal fixation.* Some fractures are extremely unstable and cannot be adequately controlled by any form of external fixation or by traction. This may be because of the planes of the fracture surfaces, the forces to which they are subjected by muscle tone or the size of one of the fragments, which may be too small to be subjected to adequate external pressure.

In these circumstances it is necessary to fix the fracture by some mechanical device applied directly to the bone. Formerly the technique of internal fixation was much hampered by the types of material available, and many different substances were employed. The introduction of stainless, non-ionisable metals removed most of these difficulties, however, and now many different techniques are employed. Trifin nails, screws, plates and screws, intramedullary nails of various types, bone grafts either fixed with screws or inlaid into slots and bone pegs of various types each have their use and applications, and new techniques or modifications of existing methods are constantly being evolved. (*Figure* 6).

Internal fixation is not only employed in unstable fractures. If surgical exposure is necessary to effect reduction, or because of delayed union or established non-union, this procedure is combined with internal fixation of the fracture by the most appropriate means.

In spite of the many effective techniques available, immobilization of a fracture by internal fixation alone is only employed in special circumstances when any form of external fixation is contra-indicated. More commonly the internal fixation is reinforced by external splinting by plaster. In these circumstances the internal fixation preserves absolute immobility at the fracture site while the plaster protects the fixing device and the fracture from stresses and strains until bony union is complete.

Once a fracture has united solidly any metal used to fix it in its early stages serves no useful function and is usually removed at a later operation.

In spite of its obvious advantages, and in spite of its attraction from a mechanical point of view, internal fixation involves operative exposure of the fracture site with all its attendant dangers. For this reason it should never be employed if the same ends can be satisfactorily achieved by simpler methods.

3. *Preservation of function.* While a fracture is uniting function must be preserved, not only in the injured limb but in the body as a whole. This, the third principle in the treatment of fractures, is just as important as the other

two and its neglect can completely mar the final results even if firm union in normal position has been achieved by scrupulously accurate reduction and subsequent effective immobilization.

Formerly the preservation of function was ill understood and much neglected. When a fracture had been 'set' and 'splinted' the surgeon's task was considered to be at an end and this attitude led to much disability which might have been prevented. Indeed this was so obvious that some schools of thought advocated the abandonment of reduction or fixation in favour of early movements, active and passive, massage and other forms of physiotherapy in an attempt to preserve function while the fracture was left to look after itself and heal naturally. During the last fifty years the question of function has received much more attention and is much better understood. It is now generally accepted that the three basic principles of reduction, immobilization and preservation of function are of equal importance and are compatible, indeed complementary, in the treatment of any fracture.

During treatment, the preservation of activity is the concern of all those who come into contact with the patient, The general management may be under the control of the surgeon, but all concerned must assist and encourage and a general atmosphere of informed enthusiasm is vital. It cannot be too often stressed, however, that enthusiasm must be informed, an over-exuberant approach can do far more harm than good. Moreover, the preservation of function is quite a complex matter, requiring not only a sound knowledge of the principles involved but also a good deal of common sense in the application of these principles to the individual patient.

There are two main causes of disfunction after a fracture. Firstly, the actual damage to bone and soft tissues produces function loss. This damage must be allowed to heal, traumatized tissues must be protected from stresses and rested while healing is taking place, and any excessive activity involving damaged structure does harm rather than good, particularly in the early stages. At the same time, local injury is associated with oedema extending far beyond the area of actual damage. If the effusion so formed is allowed to persist and organize, the resultant fibrous adhesions bind the tissues together and cause stiffness. Such oedema is best dispersed by active muscular contraction.

The second cause of loss of function is disuse, which with its associated immobility, produces changes in all the structures involved. Muscles waste and the ordinary wasting of disuse is speeded up in the presence of injury. Immobilized joints become stiff and this stiffness is more marked if there is any effusion within or around the affected joint with subsequent adhesion formation. All these changes are prevented by activity. It is true that im-

mobilization is necessary to allow healing of the broken bone, and that this is usually extended to include the joints above and below the fracture: but the fact that part of a limb is immobilized does not mean that functional activity cannot be preserved. The joints both proximal and distal to the splinted area can be exercised by active movement, and muscles within the splinted area can be kept active either by exercising unsplinted joints or by static contraction; and even immobilized joints are affected by contraction of the muscles and movement of the tendons passing over them. Moreover, the circulation is maintained by muscle contractions and use of the limb as a whole.

The key to the prevention of disuse changes while a fracture is uniting is, therefore, regular active exercises of the unimmobilized joints, static contraction of the muscles, as normal use as possible of the injured limb as a whole and regular use and exercises of the rest of the body. These exercises start as soon as possible after injury and are carried out regularly. Usually one session of exercises under supervision daily is necessary but apart from this the patient should do prescribed exercises by himself for five minutes every hour during the day.

These exercises are graduated; starting simply they become more strenuous and are carried out against increasing resistance as recovery progresses. The difficulty is, of course, that while such exercises are the ideal way to prevent disuse changes they cannot always be carried out freely because of damage to soft tissue. If activity causes further damage or strains the fracture site, it does more harm than good and is contra-indicated. To decide when exercises are or are not beneficial and how vigorously they may be pursued is a matter requiring much experience. The two cardinal signs to be considered are pain and muscle spasm. If active movement causes pain, as opposed to discomfort, the structures involved require rest. If muscle spasm is present the muscles should be rested; and any joint in which movement is prevented by muscle spasm requires immobilization and rest rather than exercise. Similarly exercises should never be pushed to a point where they produce pain or spasm, and the appearance of either of these indicates too vigorous activity. Movements should always be active, that is they should be carried out by the patient. Passive movement is only permissible under very special circumstances. In the presence of a traumatic paralysis of the flaccid type the power of active movement is lost but in the early stages uninjured joints will have a full range of free movement. Unless movements are maintained these joints will become stiff and in these circumstances it is desirable that all unimmobilized joints should be gently put through a full range of passive movements three or four times a day. Apart from this one special circumstance

passive movements are dangerous and may do much harm. Formerly this danger was not appreciated and physiotherapists and others were commonly taught to 'stretch' stiff joints. If any real increase in mobility is achieved in this way it can only be done by tearing the adhesions which limit movement. This tearing produces muscle spasm and is followed by a traumatic effusion which, when it organizes, will form more adhesions. Repeated stretching thus sets up a vicious circle of spasm, effusion and more adhesions and usually the final result is an increased stiffness of a particularly stubborn type. Active movement, on the other hand, does much less damage. Any adhesions present are stretched gradually, without widespread tearing, and stiffness is overcome very slowly.

Measures other than active exercises are of value in the prevention of disuse changes but these are of rather less importance than is sometimes supposed. The basic purpose of all such measures is the promotion of as normal a use as possible of the injured and immobilized limb. Walking instruction, occupational therapy and many of the other activities practised at rehabilitation centres encourage use of the injured limb and the body as a whole and are to this extent highly beneficial. The danger is, however, that patients may achieve their mobility not by overcoming their disabilities but by finding a way round them, by developing trick movements, abnormal gaits, exaggerated use of uninjured joints and so on. For this reason such treatment, although more spectacular, has less solid worth than graduated active exercises specifically designed to restore function to damaged muscles and joints and to prevent disuse changes in associated structures which are not directly involved in the injury.

When the fracture has united and immobilization is no longer necessary, the patient should already be well on the way to recovery. Treatment is continued on the same lines, the emphasis being on active exercise, and, providing the patient co-operates, ultimately residual disability can be reduced to that predetermined by the nature of the original injury.

This aspect of the treatment of fractures is the concern of many people, and all those who come into contact with patients suffering from injuries must understand the importance of functional activity. The mental attitude of the patient is of the greatest importance, a powerful determination to overcome disability is vital and this can only be preserved by an atmosphere of encouragement. The immediate reaction of the normal person to injury is the desire to become well again and this desire must never be allowed to fade into apathy.

TIME FACTOR

One of the first questions asked by any patient who has sustained a fracture is 'how long?'—'how long in plaster?'—'how long before I can work?'—'how long before I can play football—or golf—or tennis?' and so on.

Although there are fairly wide variations from the average, and although various complications already discussed may delay healing it is possible to estimate the period of immobilization necessary and the total period of disability with reasonable accuracy in most individual fractures. At the same time the surgeon should be careful to make it clear that recovery is not simply a question of days or weeks but depends on the healing of damaged bone and soft tissue. The answer to the question 'how long will I be in plaster?' must be 'until the bone has healed' and not so many weeks or months. The surgeon can then explain what the average time necessary for union of that particular fracture may be; but the patient must understand that it is union, not time, that matters. The period of total or partial disability can be related to the time necessary for union.

Some reference has already been made to the actual time it takes a broken bone to unite solidly and the circumstances which influence healing. The efficiency of treatment is probably the most important factor. The vascularity and the nature of the bone involved are very important. The age of the patient must be considered, fractures in children unite more rapidly than those in adults and the younger the child the more rapid is union. This difference disappears in the early teens, and thereafter the average time of union is fairly constant, being little affected by increasing age.

The time necessary to allow healing varies greatly in different bones and at different ages. For example, a fractured femoral shaft, if all goes well, in a baby will unite firmly in about four weeks, in a child of five in about eight weeks and in an adult in between twelve and sixteen weeks. With the possible exception of the phlanges, the minimum period of immobilization necessary for a fracture in an adult is about six weeks; and for some bones, for example the shaft of the femur or a tibia and fibula, six months is not by any means unusual.

The only way in which union may be speeded up is by strict attention to the basic principles of treatment already enunciated. Any attempt to cut corners will inevitably lead to disappointment if not to disaster.

SPECIAL PROBLEMS IN ATHLETES

The restoration of full function in as short a time as possible is the aim of treatment of any fracture. This is true of an athlete as of anyone else only, as it

were, more so. The difficulty is twofold. First, a small residual disability which would be of little importance in any ordinary occupation, can end the career of a first-class athlete. Secondly the time factor is often a matter of urgent concern; for the athlete the period of disability is one of enforced inactivity. Both these factors must be borne in mind by the surgeon. He must consider the nature of the sport in which his patient engages and must know something of the physical demands and the mechanics of this sport. He must take advantage of his patient's youth, physical fitness and desire to regain his former abilities. At the same time he must remember that there is no quick or easy way; the three principles of treatment still hold good and must be scrupulously observed. A bone does not unite more quickly because the patient is young, healthy and a first-class athlete.

3

FRACTURES OF THE DIFFERENT BODY REGIONS

IN THE following sections no attempt has been made to consider in detail the multitude of different fractures which may occur in the human body. This information is available in the standard medical text-books on fractures, the more advanced of which are works of some magnitude.

What has been done is to indicate the more common fractures which may occur in the course of outdoor games and sport and to give some idea of their significance from the point of view of the treatment necessary, period of disability to be expected and ultimate effects on function. Little reference has been made to the fractures and epiphyseal injuries of childhood, to the fractures of old age or to the very complicated problems which may be presented by multiple fractures. Bony injury directly involving the joints is dealt with in the section of the book devoted to joint disorders. Fatigue or stress fractures have been considered in more detail, however, and a separate chapter has been devoted to these injuries because they are common in athletes and do not always receive due attention in the text-books.

FRACTURES OF THE FOOT

Fractures of the various bones of the feet are relatively common in athletes. They vary widely in their severity and in the ways in which they are produced.

The phalanges of the toes may be broken either as a result of an unexpected contact between the unprotected foot and a hard object (a classical accident is to kick a bag of paper which unexpectedly contains bricks), or as the result of crushing when something heavy is dropped or falls on the toes. Adequate immobilization in a rigid dressing, such as gauze soaked in collodion, is the only treatment usually necessary but these fractures are surprisingly painful and this, together with the difficulty in providing suitable footwear to accommodate the splinted toe, results in an unexpectedly long period of disability.

Metatarsal fractures are not uncommon. Fractures of the neck of the bone, often multiple, are produced by indirect violence, and the maintenance of accurate reduction of this type of fracture may involve operation and internal fixation. The metatarsal shafts and bases are fractured by direct violence, the

first and fifth bones being most exposed to injury. Reduction and adequate immobilization in a below knee walking plaster is the usual treatment. The base of the fifth metatarsal is commonly fractured either by direct violence or by traction through the peroneus brevis tendon. Displacement is minimal, but firm union does not take place for between six and eight weeks. Various methods of immobilization are practised, of these a below knee walking plaster is probably the most efficient.

In the mid-tarsal region fractures of the cunieform, tarsal scaphoid and the cuboid are of importance mainly in relation to the associated damage to the mid-tarsal joints. Simple fractures without joint involvement unite readily and without subsequent disability. On the other hand, if the joints are disorganized subsequent degenerative changes are usual with mid-tarsal stiffness and pain. Severe damage may necessitate subsequent or, occasionally, immediate surgical arthrodesis of the joints involved.

Fracture of the astragalus is a high velocity injury of some rarity. Many varieties of fracture or fracture-dislocations may occur and although some of the simpler of these are relatively benign most require complex treatment and are associated with subsequent disfunction of the ankle and subastragaloid joints, which may be severe.

The Os Calcis is one of the commoner sites of fracture in the lower limb. Apart from the comparatively rare avulsion fractures when part of the bone is torn off by the tendo achilles, these injuries are due to direct violence almost invariably associated with a fall from a height on the heels, the knees and hips being extended on impact. Os Calcis fractures are usually comminuted, and in more than 60 per cent there is damage, direct or indirect, to the posterior subastragaloid joint. Fractures of the Os Calcis have acquired an evil reputation; and it is often said that when the subastragaloid joint is involved subsequent disability is inevitable and severe. The outlook is not necessarily so gloomy, however, even after subastragaloid damage many patients have no residual discomfort or disability provided full movements of all the other joints in the foot have been preserved by treatment. The management of fractures of the Os Calcis is a matter of some controversy and two main schools of thought exist. Some surgeons go to great pains to reduce the fracture accurately, manipulating the bone fragments by means of pins and applying strong lateral compression. When they have achieved as accurate reduction as possible the foot is immobilized in a plaster which incorporates the pins, and immobilization is continued until the bone is united. Others do not consider that reduction of the fracture is of major significance, and believe that maintenance of function of the rest of the foot is the vital factor. Patients are confined to bed and the gross bruising and swelling of soft tissue which is

a feature of these injuries is controlled by pressure bandaging. Exercises of the ankle, toes and mid-tarsal joints are started at once and practised assiduously. When all swelling has subsided, patients are allowed to get about on crutches, but weight bearing is not permitted until union has occurred. On the whole the results suggest that preservation of foot movement has more influence on the ultimate result than reduction of the comminuted bone and this is probably because it is impossible by any form of manipulation to restore the articular surfaces of the Os Calcis to normal and, in any circumstances, the widespread contusion of the articular cartilage of the subastragaloid joint, which occurs at the moment of injury, is inevitably followed by degenerative changes.

FRACTURES OF THE ANKLE

With the sole exception of the region of the wrist, fractures occur around the ankle more commonly than anywhere else in the body. The reason for this lies in the bone structure of the joint and the forces to which it is subjected. The upper articular surface of the astragalus, convex in the antero-posterior plane, lies in contact with the lower articular surface of the tibia which is concave to receive it. It is maintained in this position by the malleoli on each side, the medial tibial malleolus being somewhat shorter than the lateral, which is the lower end of the fibula. Areas on the sides of the astragalus are covered with articular cartilage as are corresponding areas on the inner surfaces of the malleoli and there is close bony contact at these points so that lateral movement of the astragalus in the malleolar mortise cannot take place. Posteriorly the joint is strengthened by a downward projection of the lower end of the tibia, sometimes referred to as the posterior tibial malleolus. Three of the several ligaments stabilizing the joint are particularly powerful; and the medial ligament of the ankle (the deltoid ligament) passing from the tip of the malleolus to the astragalus and Os Calcis, the lateral ligament, a tribanded structure joining the tip of the fibula to the astragalus and Os Calcis, and the inferior tibio-fibular ligament which binds the lower fibula to the tibia. (*Figure* 1). Movement between the astragalus and the tibia can occur in one plane only, producing dorsiflexion and plantarflexion of the foot. The upper articular surface of the astragalus is slightly broader anteriorly than posteriorly, a circumstance which tends to prevent a slipping forward of the tibia and fibula in relation to the astragalus.

The stresses to which the ankle is subjected vary widely in direction. The act of standing, walking and running involves compression, abduction, adduction, external rotation and internal rotation strains, usually in combination of

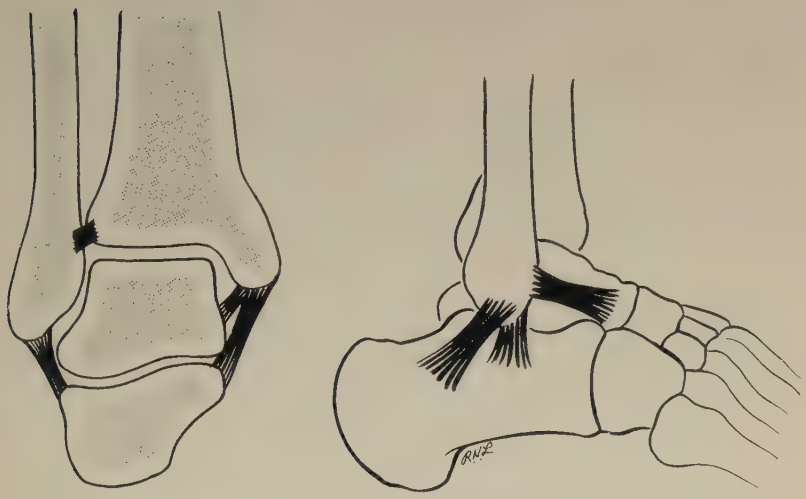

FIG. 1. *The ankle joint.* The astragalus fits into the mortise made by the lower ends of the tibia and fibula to form the ankle joint. This joint is reinforced by three powerful ligaments, the inferior tibio-fibular ligament binding the fibula to the tibia above, the deltoid ligament on the inner side and the tribanded external ligament on the outer side.

two or three at a time. Strenuous activity increases the magnitude of these forces as does inequality in the surface of the ground and a sudden sharp increase may follow accidents of various kinds.

The picture is, therefore, of a joint at which movement is possible in one plane only and of which the integrity depends on two relatively small bony projections. This joint is subject to powerful and varying stresses in many planes, most of which are resisted by bone only without any buffering by joint movement or muscle action. It is not to be wondered at, therefore, that fractures in this region occur frequently.

There are many different types of fractures in the region of the ankle and many different classifications of these injuries have been made since Mr Percival Pott, a London surgeon, included a description of them in his work on Fractures and Dislocations, published in 1768. In the main such classifications are based on the nature of the forces producing the various fractures and indeed the sub-division of this complex group of fractures can be continued almost indefinitely.

From the practical point of view, however, all these fractures except two

may be divided into two large groups, and this classification provides the key to their gravity.

The first group consists of those fractures in which normal relationship between the astragalus and tibia is maintained and stable, that is, there is no dislocation or tendency to dislocation of the ankle joint. Such fractures are usually not displaced, although the medial malleolus is sometimes separated from the tibia and may even require open reduction. Even so, all are benign, after reduction, if this is necessary, immobilization in a below knee walking plaster for between six and eight weeks is the only measure required to produce union and subsequent function of the ankle joint is normal.

In the second group the relationship between the astragalus and tibia is abnormal or unstable, in other words there is a dislocation or potential dislocation of the ankle. The degree of displacement of these fractures differs enormously, varying from a slight displacement of one malleolus with widening on the malleolar mortise and consequent lateral instability of the astragalus, to fracture of all three malleoli with wide displacement and complete dislocation of the astragalus in relation to the tibia. The principle of treatment of all such injuries is the same, however, whatever their degree. Reduction of bony displacement must be accurate, the astragalo-tibial relationship must be restored to normal, and this position must be maintained absolutely until union is completely sound, involving immobilization for some three or four months. Subsequently a further period of treatment is necessary before the function of the ankle returns to normal, and late development of degenerative joint changes is not uncommon. Reduction of these fractures is not always easy. Most surgeons rely on manipulation but in certain circumstances open reduction and internal fixation is necessary. Whatever the method employed reduction must be complete if subsequent disability is to be avoided. An accurate reduction is only the first step, even when reduced these fractures are extremely unstable and re-displacement is common. Immobilization must, therefore, be effective. Plaster must be applied with firm pressure in the ankle in the appropriate planes; X-rays must be taken at regular intervals so that if re-displacement occurs it can be detected and corrected at once; and weight bearing is not permitted in plaster until reduction to normal has been maintained for between six and eight weeks. Obviously, therefore, injuries of this second group are grave, treatment is difficult, the period of disability is longer and the danger of subsequent disfunction greater.

Two types of injury are not included in either of the groups already discussed.

Compression fractures of the ankle occur when the astragalus is driven upward into the inferior surface of the tibia, with comminution and displace-

ment of the lower articular surface of this bone. Because of the comminution, completely accurate reduction is often impossible and subsequent irregularity of the tibial articular surface and degenerative changes in the ankle joint are inevitable. Indeed, the immediate damage to the joint is so severe that it often seems that crippling disability is inevitable, and the surgeon may be tempted to carry out immediate arthrodesis. Surprisingly, however, disability after this injury is less than might be expected, and although the ankle joint is permanently damaged the disfunction produced is only moderate and the joint may be free from pain.

The last of the ankle injuries, a tibio-fibular diastasis, is not a true fracture but a disruption of the inferior tibio-fibular ligament with consequent separation between the two bones and widening of the malleolar mortise. If uncorrected, such widening would allow lateral movement of the astragalus with associated disfunction; and close contact between the bones must be re-established and maintained until the ligament is healed. This is done either by the application of plaster with firm lateral pressure over the tibia and fibula or by operation and temporary fixation of the bones with an encircling wire or some such device. If ligamentous healing without widening of the mortise is achieved no subsequent disability follows this injury.

FRACTURES OF THE FIBULA

Fractures of the lower end of the fibula have already been discussed in connection with the ankle joint. Fractures of the fibular shaft without an associated tibial fracture do occur from time to time as the result of direct violence. Subsequent displacement at the fracture site is rare, and the only complication is occasional damage to the external popliteal nerve in association with fractures of the upper third of the bone. Nerve damage results in some sensory loss over the front of the ankle and dorsum of the foot and weakness or complete paralysis of the anterior tibial and peroneal muscle groups with consequent weakness or complete loss of dorsi-flexion and abduction of the foot.

The fibula is not a weight-bearing bone and the only stresses to which it is habitually subjected arise from contraction of the muscles around it. Immobilization in plaster is not, therefore, necessary in isolated fractures of the fibula shaft, which are treated as soft tissue injuries. Associated damage to the external popliteal nerve requires the treatment appropriate to such nerve injuries.

FRACTURES OF THE TIBIA

Fracture of the medial malleolus has already been discussed and fractures of the upper end of the tibia involving the condyles and upper articular surfaces, a very important group of injuries, are dealt with in the section devoted to injuries of the knee joint.

The fractures of the tibial shaft without associated damage to the fibula do occur as the result of direct or indirect violence. In the main these fractures present the same problems and require the same treatment as those with associated fibula fractures, which are discussed in the following section. The presence of an intact fibula tends to limit displacement at the tibial fracture and to reduce instability, making treatment considerably simpler than is the case when both bones are broken.

FRACTURES OF THE TIBIA AND FIBULA

Fractures of the shafts of the tibia and fibula occur fairly commonly in all age groups, being produced by both direct and indirect violence. When due to direct violence both bones are usually broken at the same level in the transverse plane; and high-velocity injuries commonly cause extensive comminution or even double fractures of the tibial shaft. Indirect violence produces oblique fractures, and the level of bony injury is different in the two bones, the fibula tending to break in its upper third and the tibia about the junction of its middle and lower thirds. The tibial damage is usually more extensive than appears at first sight, with oblique cracks extending into the bone well away from the level of the fracture.

Because the antero-medial surface of the tibia is subcutaneous many of these fractures, particularly those produced by direct violence, are compound.

The basic principles of treatment are the same in all these fractures. Reduction must be accurate, and although complete apposition of the fractured surfaces is desirable the presence of any angulation or rotatory displacement is a more important source of subsequent disability. Immobilization must be completely effective, these fractures, particularly those in the oblique plane, tend to be unstable. Re-displacement is common and delay in union is certain if any movement is permitted at the fracture site after reduction. The usual method of treatment is reduction by manipulation and subsequent immobilization in a full length plaster extending from the upper thigh to the metatarsal heads. For many reasons, however, it may be necessary to perform an open reduction of the tibia and fix the fracture or fractures with metal plates and screws, intramedullary nails or some other mechanical device. The tibia is a

subcutaneous bone and easily accessible and such operations present obvious attractions because of the accuracy of reduction and subsequent stability which can be achieved thereby. The penalty for failure, and in particular the penalty for any subsequent wound infection, is high. Such infections spread quickly to the adjacent fracture where they are associated with indefinite delay of union and often with extensive loss of bone at the fracture surfaces. These complications involve prolonged treatment, often for a period of years, and may even result in amputation of the leg below the knee. For this reason operation should never be undertaken lightly and without good reason.

The tibia is the second largest long bone in the body and however perfect the reduction and effective the subsequent immobilization union of a fracture of the tibial shaft takes some time. Firm union is not to be expected in an adult in under three months and the average period of immobilization necessary is probably between four and five months and in some instances may be longer. During this time, toe exercises and static muscle contraction must be practised vigorously. Later a course of graduated exercises is necessary to restore knee, ankle and foot movement and muscle bulk and power, so that the total period of material disability following a fracture of the shaft of the tibia and fibula in an adult is seldom less than six months. Nevertheless, with adequate treatment, and provided no untoward complications arise, full function is normally to be expected after these injuries. Commonly anxiety is expressed lest the prolonged immobilization necessary may cause permanent stiffness of the knee and ankle joints. Curiously enough, most attention is directed to the knee whereas in fact this joint, being above the fracture, is less likely to be affected than the ankle which, being below the fracture, will be involved in any oedema which follows soft tissue injury and vascular damage at the fracture level. Provided adequate treatment on the lines already indicated is carried out during the period in plaster, immobilization, however prolonged, is not itself responsible for permanent limitation of movement of either the knee, ankle or foot.

FRACTURES OF THE PATELLA

Opinions of the value of the patella as an integral part either of the knee joint or the extensor apparatus of the knee, the quadriceps mechanism, have changed considerably during the past twenty-five years. Formerly this bone was considered a vital structure and much was made of its function as a sesamoid bone, as a device for preventing friction between the quadriceps and the lower end of the femur, as a means of holding the quadriceps away from the front of the knee joint and thereby providing better leverage for this muscle

and so on. More recently, however, considerable doubts have been cast on the value of a patella and there is good reason to believe that it is in fact a retrogressive structure in fast-moving mammals. Certainly the bone can, when necessary, be removed completely and provided the defect in the quadriceps is adequately repaired no loss of function of the knee joint follows.

Fractures of the patella are fairly common in adults and may well occur in the course of games and sports. When dealing with these injuries the essential consideration is whether or not they are associated with disruption of the quadriceps mechanism as a whole; and this is mainly determined by the type of violence producing the injury.

When broken by direct violence, the patella is crushed against the underlying femoral condyles and the fracture produced is stellate or comminuted. The integrity of the extensor mechanism is not disturbed, and, as far as the fracture is concerned, the only treatment necessary is immobilization with the joint in extension for a period of between six and eight weeks. The measures necessary to deal with the associated haemarthrosis are discussed in the section dealing with the knee joint; and subsequently a course of graduated exercises is necessary to restore the muscle bulk and power and the joint movement. One late complication of this type of fracture may occur; damage to the articular cartilage of the patella and femoral condyle may be followed by development of patello-femoral arthritis, which may make late excision of the whole bone necessary.

The patella can also be broken by indirect violence transmitted to it by the quadriceps muscle. In this type of injury the fracture line is transverse, separation between the fragments is usual, and the bony damage is associated with transverse tearing of the fascial expansion of the quadriceps. These tears extend laterally across the knee joint in the line of the fracture and may be extensive. The essential feature of treatment is surgical repair of the quadriceps followed by immobilization of the joint in extension until the muscle has healed soundly, a process taking some six or seven weeks. The fascial tears are sutured directly but methods of dealing with the fractured patella differ with the type and site of the fracture and the personal preference of the surgeon. Formerly, when this bone was considered important, it was customary to repair the fracture by suturing or wiring the fragments together. Today it is more usual to excise either one or both patellar fragments and suture the quadriceps tendon in such a manner as to obliterate the resultant defect. After effective repair and sound healing a course of exercises is necessary to restore full function of the muscle and joint but normally no material residual disability should occur.

FRACTURES OF THE FEMUR

The femur is the largest and strongest bone in the body and can withstand very considerable force of all kinds. Femoral fractures in adults only occur as the result of severe injuries in which the mechanical forces involved are great. In old age the upper end of the bone becomes progressively weaker and fractures in this region are common.

Fractures of the lesser trochanter occur occasionally in the course of games, particularly in schoolboys, in whom the epiphyseal line is not fused. The injuries are of the avulsion type, force being transmitted from the psoas tendon. Displacement is slight, reduction or immobilization unnecessary and a period of rest until acute symptoms disappear is the only treatment necessary. The greater trochanter is also occasionally fractured either by muscle action or direct violence. Again displacement is slight and a suitable period of rest is all that is indicated. Fractures of the femoral neck are injuries of the aged and the many problems associated with these fractures are outside the scope of this work. The same applies to the other fracture of old age in this region, the per or inter-trochanteric fracture.

Fractures of the femoral condyles are discussed in connection with the knee joint.

Fractures of the femoral shaft occur from time to time as the result of accidents in which powerful forces are involved. In the middle half of the shaft these can be reduced by traction and immobilized by a combination of traction and external fixation in a splint of the Thomas type.

Fractures of the upper and lower regions of the shaft present special problems because of difficulty in aligning the short upper or lower fragment with the main bone. The present tendency is to employ open reduction and internal fixation with an intramedullary nail of the Küntscher type to control and immobilize these fractures and this method can also be used, when necessary, in any shaft fracture.

If reduction and immobilization are adequate, fractures of the femoral shaft in adults unite quite readily. The average period of immobilization necessary is between twelve and sixteen weeks. During this time, static quadriceps contraction and exercises of the unsplinted joints are practised. Several of the methods of traction in common use allow a limited range of both hip and knee movements to be carried out during the period of immobilization. When the fracture has united, the usual period of rehabilitation and exercises is necessary to restore full function of the limb. In a young and healthy adult in whom reduction has been accurate and union sound, the fracture of the femoral shaft should produce no permanent disability.

FRACTURES OF THE PELVIS

The bones making up the pelvis form a massive ring which serves as a scaffolding for the transmission of body weight and other forces from the lower spine to the femoral heads. It also forms a protective shield for the pelvic viscera and affords large bony surfaces for the attachment of the muscles of the abdominal wall, the pelvic diaphragm, the hip and the thigh. Weight and stress transmission occur mainly through the posterior half of the ring where the pelvic bones are much more massive than in front.

A few avulsion fractures of the various processes may occur in the course of games, being seen mainly in teenagers in whom these processes have not yet fused to the main pelvis. The anterior superior and anterior inferior spine of the ileum and the ischial epiphysis may be detached by muscle action but displacement is slight and a period of rest the only treatment necessary.

The sacrum may be fractured by a direct blow. The displacement at the fracture line is slight or absent, immobilization is unnecessary and rest in bed until symptoms disappear is the treatment indicated.

Fracture of the coccyx is not uncommon as the result of an unexpected fall in a sitting position. Angulation at the site of the fracture is usual, but reduction is unnecessary, and effective immobilization is not possible. These fractures do not cause any material disability but initial discomfort is considerable and is sometimes surprisingly persistent, pain on sitting being present for months. Complete removal of the fractured fragment may be necessary as a late measure if symptoms are unduly persistent, but the results of this procedure are not as uniformly satisfactory as might be expected.

FRACTURES OF THE PELVIC RING

Fractures of the pelvic ring as a whole are caused by crush injuries such as might occur when a rider is rolled upon by a fallen horse. These fractures fall into two different classes.

Fractures in which the ring as a whole is not disrupted are usually single and most often occur in the pubis, the weaker anterior part of the pelvis. Displacement does not occur; there are no important associated injuries and the ring remains stable. Rest until the acute symptoms subside is the only treatment necessary.

Fractures with disruption of the ring are much more serious injuries. Such fractures are usually double, the most common type being a fracture of the pubis anteriorly and the ileum in the region of the sacro-iliac joint posteriorly. Other varieties include a single fracture and a disruption of the symphysis

pubis or one sacro-iliac joint or a double fracture of the pubis. These injuries are usually associated with considerable displacement of part of the pelvic ring, usually carrying the hip joint and consequently the whole leg with the displaced fragment. Reduction must be effected by traction or manipulation and immobilization by traction in various planes or by means of a plaster spica and must be maintained for three or four months until union has taken place. Internal injuries, usually to the bladder or urethra, are not uncommonly associated with disruption of the pelvic ring and must, of course, receive appropriate treatment. This type of pelvic fracture is a severe injury and some degree of residual disability is usual.

4

FRACTURES OF THE DIFFERENT BODY REGIONS (Contd.)

FRACTURES OF THE ARM

ALTHOUGH the arms do not have to sustain the weight-bearing stresses to which legs are subjected and are only indirectly concerned in locomotion, the multitude of their other functions involve more risk of accidental injury. The bones of the arm are also smaller and less strong than those of the leg and for these reasons fractures occur more frequently in the upper limb than in the lower. This generalization holds good in relation to injuries sustained in the course of games and sports.

Fractures of the phalanges

The bones of the fingers are comparatively slender and these digits are very liable to injury so that phalangeal fractures are relatively common. Fractures of the shafts of the middle and proximal phalanx are usually angulated and must be reduced and immobilized with the fingers in flexion in the correct plane. Major fractures involving the interphalangeal joints must also be reduced with care and immobilized in flexion. The phalanges unite quickly; splinting for three or four weeks is usually sufficient, and subsequently a course of wax baths and finger exercises helps to restore full mobility. Unless finger fractures are associated with gross soft tissue damage or extensive injury to the interphalangeal joints, they should produce no permanent disfunction. Unfortunately, much avoidable disability occurs because such fractures are regarded as minor injuries and insufficient care is taken in their management. The most common errors are inaccurate reduction or immobilization of the injured digit in extension, both of which are certain to be followed by some degree of stiffness.

Fractures of metacarpal bones

A large variety of fractures of different types may occur in the metacarpals. In general there are three main varieties, fractures of the neck, the shaft or the base, but the incidence of these is not equally distributed among the five bones. Fractures of the neck occur most often in the fifth metacarpal although

they are sometimes seen in the second, third and fourth. They are caused by direct violence, usually by striking something hard with the closed fist. Posterior angulation at the fracture site is almost uniformly present, and unless this is corrected some permanent abnormality of metacarpo-phalangeal movement results. Reduction can only be achieved and maintained with the metacarpo-phalangeal joint in full flexion and failure to appreciate this point is often the cause of permanent deformity. Fractures of the shaft of any metacarpal may occur as the result of either direct or indirect violence. Gross displacement is unusual, and immobilization in a below elbow plaster, which should include the flexed finger corresponding to the fractured metacarpal, for about six weeks is the treatment indicated. Fractures in the region of the bases may occur in any metacarpal but they are by far most frequent and most important when they affect the first metacarpal. Two types of fracture occur in this region. In one, the articular surface of the bone is not involved and the trapezio-metacarpal joint remains intact. Such fractures are treated by manipulative correction of any angulation which may be present and immobilization in plaster. In the second and more serious, a compression force in the long axis of the bone produces an oblique fracture of the base, involving the articular surface. The smaller fragment remains in position, while the remainder of the bone, carrying with it the thumb, dislocates in relation to the trapezium. This fracture-dislocation, associated with the name of Bennett, can only be reduced and controlled by traction; and to be effective this traction must be in the correct plane. A plaster incorporating a metal strut with a curved end which extends beyond the tip of the thumb is applied to the hand and forearm. Traction between the end of this strut and the thumb is arranged by means of a pin transfixing the pulp of the thumb and a suitable elastic band. The thumb must be pulled on in the correct plane, that is in the direct line of the shaft of the radius. Far too often the whole method is rendered useless because the traction pulls the thumb into the abducted position. If traction is in the proper plane, the fracture reduces easily and accurately; and provided reduction is maintained by continued traction union takes place in about six or seven weeks. This type of splint requires constant care and adjustment but with intelligent supervision it is possible to achieve union in normal position without any disturbance of the joint and without subsequent disability of the thumb.

Fractures in the region of the wrist

More fractures occur in the region of the wrist than anywhere else in the body, and the vast majority of these are caused by falls on the outstretched hand. The overall incidence of the different types of fractures varies with the

age groups of the patients concerned and is determined by the relative strength of the bones in this region at different ages. The same type of force, produced by a similar accident, may cause a Greenstick fracture of the lower radial shaft in a child of three, a fracture dislocation of a radial epiphysis in a child of ten, a fracture of the carpal scaphoid in a young man of twenty and a Colles type fracture of the lower end of the radius in a lady of sixty-five. Various fractures in this region are relatively common in athletes and sportsmen.

Fractures of the carpal bones

Isolated fractures of the different carpal bones do occur, by far the commonest and most important being fracture of the carpal scaphoid. The scaphoid is broken by compression forces usually produced by a fall on the outstretched hand, and the great majority of these fractures occur in young adults between the ages of eighteen and thirty. The plane of the fracture is transverse but it may traverse the proximal, middle or distal thirds of the bone. Displacement is unusual and a recent fracture is often only a linear crack so that it may be difficult to detect even in an adequate series of good quality radiographs. In many scaphoid fractures the immediate symptoms are not severe and some patients may not seek medical advice while in others the presence of a fracture may not be suspected even on examination. The blood supply of the carpal scaphoid varies and a fracture may cut off one fragment and render it avascular. If this happens, union even under the best conditions is inevitably delayed. Fractures of the carpal scaphoid have a bad reputation; it is said that the bone does not unite well and that in many instances repair will not take place at all. Basically the trouble is that many such fractures are not well treated. Some are not diagnosed at the time of injury and treatment is delayed. Even when they are diagnosed effective immobilization is not easy, the proximal and distal fragments lie in line and move with the proximal and distal rows of the carpal bones and any movement at the fracture site delays or prevents union. The wrist must be immobilized in a snug close-fitting plaster extending from the metacarpal heads to the elbow and including the thumb as far as the interphalangeal joint. This plaster must be kept in good repair and changed at once if it shows signs of becoming loose. If properly immobilized immediately after injury, most scaphoid fractures unite in between eight and ten weeks, a few in which the blood supply between the fragments has been affected require immobilization for some months but complete failure of union is rare. Non-union is not necessarily associated with severe disability, many people with un-united fractures are quite unaware of any abnormality.

Various complex fractures and fracture-dislocations of the carpus occur. Of these the more usual are dislocation of the semilunar bone, perilunar carpal dislocations, transcapho-perilunar carpal dislocations, dislocations of the semilunar and half the scaphoid, dislocations of the semilunar and the scaphoid and perilunar-scaphoid dislocations of the carpus. Other more unusual types of fracture and fracture dislocations are also reported from time to time. Essentially the treatment of all such injuries is the same; early reduction and effective immobilization. Reduction is usually comparatively simple if carried out soon after injury, but becomes progressively more difficult as time goes by. The prognosis after such injuries depends largely on how soon effective treatment is instituted.

Fractures of the lower end of the radius

In 1814 Abraham Colles published a paper on 'The Fracture of the Carpal Extremity of the Radius' in the *Edinburgh Medical and Surgical Journal;* and ever since the fracture described, the commonest in the body, has borne his name. Although it occurs most often in middle and old age, a Colles' fracture is not uncommon in young adults and may well occur in athletes. The classical features of this injury, a fall on the outstretched hand, the 'dinner-fork' deformity, a complex displacement in several planes of the lower radial fragment, the techniques of reduction and immobilization, the incidence of redisplacement, the various complications which may arise and the end results to be expected are fully discussed in all text-books on fractures. In young adults the lower end of the radius is less brittle than in later life and gross comminution in the region of the fracture site is rare. At this age it is usually possible to achieve a completely accurate and stable reduction, firm union is to be expected in six or seven weeks and complete functional recovery is the rule.

A Colles' is not the only fracture which occurs at the lower end of the radius. Fractures of the styloid process with or without displacement are not uncommon, and another variety is the Reverse Colles' or Smith's fracture. The basic treatment for these injuries is the same, accurate reduction and immobilization until union is firm, and good results are to be expected.

Fractures of the styloid process of the ulna

The styloid process of the ulna, usually fractured in conjunction with the radius in a Colles' fracture, may be broken without any other associated bony damage. Displacement is not material and immobilization of the wrist in a plaster slab for about six weeks is almost always followed by complete recovery.

Fractures of the radius

The radius and ulna have the same general stabilizing effect on each other as have the tibia and fibula since they are bound together by the ligaments of the inferior radio-ulnar and elbow joints and the interosseous ligament and gross displacement at a fracture of one bone cannot take place so long as the other and the ligaments remain intact.

Isolated fractures of the radial shaft are not uncommon. In the lower third 'greenstick' or complete transverse fractures occur in children and may well occur in adults. They are not usually associated with ligamentous damage and when displacement occurs overlapping cannot take place. In spite of this it may be surprisingly difficult to secure adequate apposition of the fracture surfaces by manipulation, because of the interposition of fibres of the pronator quadratus muscle. About the junction of the middle and lower thirds of the bone transverse fractures with disruption of the inferior radio-ulnar joint occur fairly frequently. Displacement and overlap are usual in these fractures. They are not easy to reduce and are extremely unstable, so that open reduction and internal fixation is often employed in their treatment. Isolated fractures of the upper third of the radial shaft without ligamentous damage are rare and when they do occur displacement is minimal. If immobilization is to be adequate, fractures of the radius must be protected against rotatory as well as angulatory and compressing strains and this involves effective immobilization of the elbow as well as the forearm, wrist and hand to the metacarpal heads.

Fractures of the radial head and neck are, strictly speaking, injuries of the elbow joint. They are caused by indirect violence acting in the long axis of the radius and impacting the head of the bone against the capitellum of the humerus. The bony injury so produced is associated with damage to the articular cartilage of the capitellum and sometimes with damage to the ligaments of the elbow and it is this associated damage which is responsible for the permanent limitation of elbow movement which sometimes follows these injuries.

Fractures of the ulna

The shaft of the ulna may be broken by direct violence, usually in the form of a blow, without joint or ligamentous injury. Such fractures are rarely grossly displaced and, provided they are adequately immobilized, unite without subsequent disability.

Displaced fractures of the ulnar shaft occur in association with dislocation of the head of the radius. These may be extension injuries, when the head of the radius is dislocated anteriorly, the so-called Monteggia fracture and, much

less usual, flexion injuries with posterior dislocation of the radial head, the reverse or flexion Monteggia fracture. The management of these extremely difficult and complex injuries is outside the scope of this work, and even in expert hands they are often followed by some permanent disability. Fractures of the upper end of the bone, the olecranon process, are produced by traction through the triceps. A fairly common injury, this fracture is associated with loss of extension of the elbow and, if the avulsed fragment is sufficiently large, disturbance of the hinge mechanism of the elbow and a tendency to anterior dislocation of the joint. Un-displaced olecranon fractures may be immobilized in extension. If displacement is present the olecranon fragment is too small to be controlled by manipulation and must be sutured back into position or, if it is sufficiently small, excised and the triceps sutured directly to the ulna. Provided repair and healing are adequate, full mobility of the elbow and normal power of extension are to be expected.

Fractures of the radius and ulna

Fractures of the shafts of the radius and ulna may be produced either by direct or indirect violence. Displacement, often gross, is usual and there may well be extensive associated soft tissue injury. Satisfactory results following these fractures depend on completely accurate reduction of both bones, and this is not easy to achieve by manipulation. Even when reduced these fractures are extremely unstable so that re-displacement is common. For these reasons, open reduction and internal fixation of both fractures, usually with plates and screws, is often necessary. Fractures of both bones must be adequately immobilized even after internal fixation and a period of about three months in a plaster extending from the upper arm to the metacarpal heads is necessary. During this time the usual exercises are practised, and when union is sound a course of further exercises for some weeks is necessary to restore power and joint movement. Provided reduction to normal has been achieved and maintained full function of the arm is ultimately to be expected.

Fractures involving the elbow joint

Fractures involving the elbow are discussed in the section dealing with injuries to this joint.

Fractures of the humerus

Fractures of the lower end of the humerus involving the elbow joint are dealt with in the appropriate section.

Fracture of the supra-condylar region of the humeral shaft occurs in children and occasionally in adolescents but almost never in adults so that these

injuries are not often seen in athletes. The difficulties associated with their management are fully discussed in any text-book on fractures.

Fractures at various levels of the humeral shaft may be produced by indirect or, less often, direct violence in adults but they are not very common. In the oblique fractures, displacement, apart from some shortening, is not usual; but if the fracture line is transverse various degrees of angulation may occur as the result of muscle action. Most of these fractures are immobilized in simple plasters, either extending from the shoulder to below the elbow, or of the shorter 'hanging cast' type which only extends from the middle of the humerus to the wrist and depends for its effectiveness on gravity. Most shaft fractures unite quite readily in about ten weeks and full function of the arm returns reasonably quickly after the plaster has been removed. From time to time, however, transverse fractures are slow to unite, and, when this happens, more complete immobilization is necessary. This can only be ensured by the use of a spica, a plaster which includes the trunk and the whole arm. When it is necessary to keep the arm in a spica of this type for some months to ensure union some residual stiffness of the shoulder and elbow is inevitable, but a course of graduated exercises is usually followed by restoration of joint movement to normal.

Adduction and abduction fractures of the humeral neck are fairly commonly caused by falls on the outstretched arm and although this is usually an injury of the elderly it may occur in the course of games and sports. If displacement is present it can be corrected by a gentle manipulation and the arm is then bound to the side and supported in a sling until the fracture has united. When union is sound and muscle spasm around the shoulder joint has disappeared a course of active exercises to restore shoulder movement is necessary.

A number of very complex fractures around the upper end of the humerus are associated with dislocation of the shoulder joint itself. The management of these injuries is difficult and the results of treatment are often imperfect. These injuries are discussed in the section of the book dealing with the shoulder.

Fractures of the scapula

The shoulder blade is a flat bone whose main purpose is to afford a large surface for muscle attachments. It is true that it articulates with the humerus to form the shoulder; but the main attribute of this joint is a wide range of movement, and the forces to which it is subjected are not sufficiently large to demand a more massive or stable bone structure.

Fractures of the scapula of various kinds do occur, mainly as the result of

direct violence. These may affect the body of the bone, the neck, when the whole of the glenoid cavity carrying the articular surfaces may be detached, or the acromion or corocoid processes. Displacement of the various fractures is usually slight and immobilization of the arm in a sling for about six weeks is all that is usually necessary.

Fracture of the clavicle

The collar bone is frequently the site of fracture, usually as the result of indirect violence produced by a fall on the shoulder. A fractured clavicle is one of the commonest of sporting injuries and its effective management is a matter of some importance.

Most clavicular fractures occur in the middle third of the bone and displacement of the fragments with overlap is usual. Reduction is easily effected by a drawing back of the shoulders and practically all the methods of immobilization in use depend on some form of brace or bandage acting on the shoulders. Not all the appliances used are equally effective by any means, and those that are must be properly applied and adjusted regularly. A good brace of the type used by Tucker not only immobilizes the fracture but permits movement of the injured shoulder while the fracture is uniting. Clavicular fractures unite readily enough and should not be followed either by disability or by cosmetic blemish.

Fractures of the thorax

Fractures of the ribs are fairly common in athletes, occurring as the result of a direct blow in the chest, one or two ribs at the most being affected by this type of injury. Displacement and internal injury are rare and effective immobilization of the chest by strapping is the treatment indicated.

More serious injury to the thorax is produced by indirect violence in crush injuries of the chest. These types of fractures are multiple and may be associated with internal injury when the sharp end of the broken ribs penetrate the thoracic viscera. Bleeding into the pleural cavity is common, producing a haemothorax. If the lungs are punctured air may escape producing a pneumothorax and if the escape is 'one way', that is if the air can leave the lungs but cannot re-enter, pressure will build up in the pneumothorax with grave consequences if unrelieved. The management of multiple fractures of the ribs with internal injuries is important but is outside the scope of this work.

Fractures of the sternum are rare injuries. A few are caused by a direct blow, but more often the sternum is involved in the multiple injuries following severe crushing.

Facio-maxillary injuries

Various injuries to the facial bones may occur in the course of games and sports and are in fact quite commonly the results of a direct blow from a ball, bat or stick. The mandible, the maxilla, the malar or the nasal bones may be fractured and different combinations of fractures and displacements are not uncommon.

The management of facial fractures, particularly of those associated with displacement, requires co-operation between the dental and plastic surgeons and is best carried out in a facio-maxillary unit where full facilities exist for the rather specialized treatment which is necessary.

5

'STRESS' OR 'FATIGUE' FRACTURES

THE ORDINARY fracture is the result of a single injury, a force in excess of its tolerance falls on a bone which then breaks, the fracture occurring at the moment of application of the violence which produces it. There is, however, another type, the so-called 'Stress' or 'Fatigue' fracture.

Both the history and nature of these injuries are interesting. In 1855 Breethaupt described a painful condition of the feet which occurred in recruits during their initial army training. This syndrome became well recognized, and with the coming of radiology it was found to be due to a type of fracture which, because of its aetiology, was called a 'March' fracture. It was recognized that one of the essential features of this injury was that it occurred as the result of repeated strains and was not caused by one single violence. As X-rays became of better quality and were universally used it was possible to correlate the clinical findings and the bone changes. It was quickly obvious that this type of fracture occurred much more often than had been supposed and affected many different bones. It also became clear that so-called 'Insufficiency' fractures and other allied conditions were really the same as these 'March' fractures of the metatarsals. At first some difficulty was experienced in distinguishing between a fatigue fracture when the callus of repair had formed and a bone sarcoma and, even after biopsy, errors were made and unnecessary amputations were performed. As increasing numbers of these injuries were reported, particularly during the two World Wars, the term 'March' fractures was gradually discarded in favour of the terms 'fatigue' or 'stress' fracture, the nomenclature now in use.

THE NATURE AND CAUSES OF STRESS FRACTURES

It is now established that Stress fractures are relatively common and occur in many different bones, but we still do not know with certainty how or why they happen or the exact nature of the forces which produce them. It is fashionable to liken them to the Fatigue or Stress fractures of metals but it has always seemed to the author that this analogy cannot be pressed too far. In metals the 'Fatigue' or 'Stress' fracture is an immediate phenomenon; a sudden linear change takes place in the internal structure of the affected metal,

which is at once rendered brittle, so that it breaks along this line as soon as it is subjected to any stress. In bone, on the other hand, there can be little doubt that these fractures occur gradually. Symptoms frequently precede demonstrable bone changes and radiographs often appear completely normal for some time after the appearance of the typical clinical syndrome. Moreover, many of these fractures are incomplete and even when they are complete displacement is unusual. By the time the fracture is visible repair, as shown by the appearance of callus, has often started.

Both the clinical and radiological characteristics of these injuries can be explained by the assumption that the affected bone is subjected to abnormal and repeated stress, causing a gradual disruption which may culminate in complete fracture. The forces causing these fractures are probably unusual. The great majority of stresses falling on the skeleton as the result of locomotion are compressing in nature and most are in the line of the long axis of the bones. The internal bone structure is built up by trabeculation to resist such forces; bones are strongest in the planes in which they most require strength. It has been pointed out, however, that during activity, and particularly during violent activity, the forces concerned with the transmission of body weight to the ground and the propulsion of the body through space are not the only ones which may affect the skeleton. Violent and repeated muscle contraction,

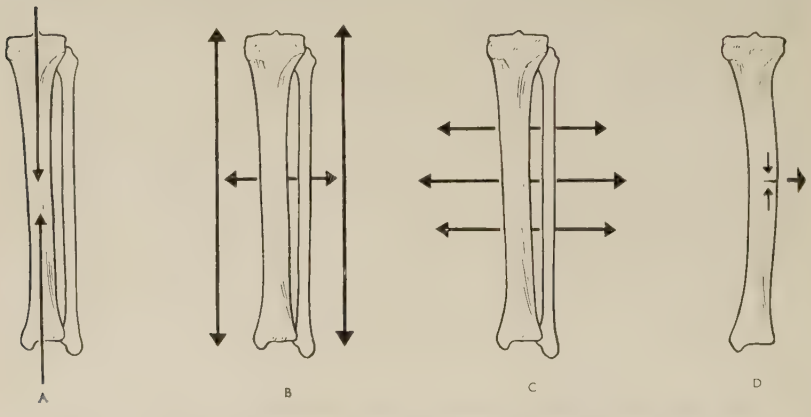

FIG. 1. *The forces producing a stress fracture.* Normally the main force falling on a long bone is compression (A). During violent activity, however, the forces produced by rapidly repeated strong contraction of the various muscle groups are added to that of gravity (B), and cause a 'whipping' movement of the bone (C). If continued for a sufficiently long time, this movement produces progressive bone damage, the so-called 'Stress fracture' (D).

particularly of the long muscles, acts on the long bones and does not necessarily produce simple compression only. Instead muscle contraction may tend to bend these bones, and repeated contraction produces a sort of whipping action, so that the bone bends to and fro. This results in repeated stretching of alternate sides of the bone; and this stretching is exaggerated if, as so often is the case, the bone is curved, when the convex surface is subjected to the greater extension strain. (*Figure* 1).

It might well be that repeated stretching of this nature causes a progressive breaking down of the trabeculae of the affected bone in the line of greatest movement. This disruption might be expected to start at the surface of the bone so that the earliest breaking down of bone would be associated with subperiosteal bleeding and, later, subperiosteal new bone. Thus repair would begin at the same time as bone disruption and, if the exciting force continues, the two processes, healing and progressive breaking down, would continue side by side. If the activity responsible for these changes is stopped the fracture may never become complete, if it is continued after the fracture is complete displacement at the fracture line may occur. If the process of repair outstrips the bony damage a state of equilibrium may be reached and no further bony damage may occur, that is to say the fracture remains incomplete. If, on the other hand, bone weakened by the onset of a fatigue fracture is subjected to too much stress it may suddenly break with a consequent dramatic change in the clinical picture. (*Figure* 2).

Some such hypothesis as this would explain some of the characteristics of stress fractures.

INCIDENCE OF STRESS FRACTURES

Stress fractures occur most often in those who undertake regular and strenuous activity. For this reason they were probably more common during the two world wars when large numbers of people took part in intensive physical training. The effects of such training were probably more marked because many of those involved were unaccustomed to exercise and some were in poor physical condition. Since the second world war many of the stress fractures reported have been in athletes, dancers, skaters, and others who lead strenuous lives. No section of the community and no age group is completely immune, however; fatigue fractures have been reported in the elderly, in children and in those who are sedentary in habit. There is no evidence to suggest that these fractures are associated with any general bone disorder or, indeed, with any other abnormality.

As one might expect from what we know of their aetiology fatigue fractures

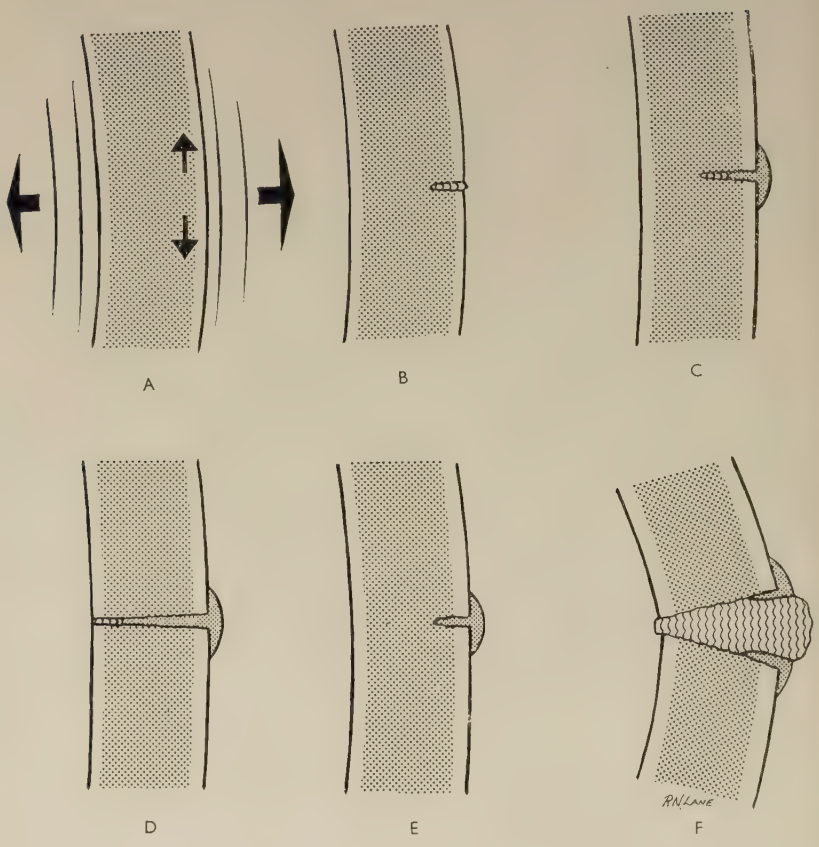

FIG. 2. *The mechanism of production of a stress fracture.* When a bone is 'whipping', the disruptive force is greatest on its convex side (A). If this movement is continued for too long some bony trabeculae give way at this point (B) and a small fracture is produced which behaves in the normal way, that is, it tends to heal. If 'whipping' is further continued, bony damage extends gradually deeper and deeper into the bone and at the same time normal healing with callus formation begins in the area first damaged (C). Very often these two processes progress side by side and by the time the fracture extends across the whole bone, healing is well advanced (D). If, however, the activity producing the 'whipping' is stopped, the fracture may not spread and local healing continues until consolidation is complete (E). Sometimes, the bone, weakened by the initial small fracture, may suddenly break completely, in which case the bony damage far outstrips the process of repair (F).

occur most commonly in the leg. The metatarsals are the most usual site but they occur quite frequently in the Os Calcis, tibia, fibula and the femur. They have also been reported in the pubis, ulna, the first rib and the tibia and fibula simultaneously.

TREATMENT OF STRESS FRACTURES

The management of a stress fracture is essentially the same as that of any other fracture, the basic principles still hold good. Reduction is not often necessary as displacement is rare, but the fracture must be immobilized and functional activity preserved until healing has taken place. If, as is often the case, the fracture is incomplete, and healing, as shown by new bone formation, is well advanced when the diagnosis is made, complete immobilization may not be necessary. It may be enough simply to stop the exciting activity and often this causes the symptoms to disappear and allows bone healing to continue undisturbed. A fatigue fracture, even of this relatively benign type, should never be underestimated however. Ample time should be allowed for consolidation and any premature resumption of strenuous activity is inevitably followed by a recurrence of symptoms and a re-appearance of bone changes.

STRESS (FATIGUE) FRACTURES OF THE METATARSALS: ('MARCH' FRACTURE)

Stress fractures were first reported in the metatarsals of recruits undergoing their initial army training, indeed the clinical syndrome was well known before the coming of radiology. There is no doubt that the most common cause of these injuries is marching or walking on hard surfaces, and for many years all the fatigue or stress fractures were called 'March' fractures irrespective of their site. Although increasing knowledge and better X-ray facilities have shown that stress fractures affect other bones much more often than was supposed the metatarsals are the commonest site of these injuries.

Site

The middle or distal thirds of the shafts of the second or third metatarsals are the most usual sites, the distal being more commonly affected than the middle third. Stress fractures can occur in any metatarsal shafts, however, and occasionally they are multiple, several metatarsals being affected at the same time.

Clinical findings

A history of some unusual activity, often marching or walking on hard ground, is associated with the gradual onset of pain of one foot. Initially the pain is not severe and is relieved by rest, but if the exciting activity is continued it becomes worse, is associated with a limp and finally becomes incapacitating. Symptoms often begin to decrease spontaneously three or four weeks after their onset. Occasionally the condition develops more suddenly and a few patients can remember the exact moment of the onset of pain. In these circumstances the initial symptoms are usually more severe and incapacitating.

On examination the patient walks with a limp and there may be a little swelling on the dorsum of the affected foot. The most important sign, however, is tenderness of the affected metatarsal shaft and if callus formation has taken place there is a palpable swelling of the bone at the fracture site.

Various foot abnormalities have been reported in association with these fractures and some observers consider such abnormalities of aetiological significance. They are not a constant finding, however, and when present may take any form.

Although the clinical syndrome is characteristic the diagnosis is confirmed by radiological examination. Here again the findings are typical, but vary according to the stage the fracture has reached. Sometimes symptoms precede bone changes and the first radiographs may show no abnormality at all. Later, the fracture may appear as a very fine linear crack which may be incomplete. Even at this stage subperiosteal new bone formation around the fracture site is quite usual. Later the fracture line may become more obvious as the result of bone absorption at the fracture surfaces, and occasionally some displacement may occur between the bone ends although this is usually slight.

Treatment

Reduction is almost never necessary but these fractures must be protected from stress. The functional activity of the foot must be preserved until the fracture has united.

In incomplete fractures or in fractures in which healing is well advanced and in which the symptoms have already begun to decline it may be sufficient to restrict the patient's activity and to instruct him in toe exercises which he should practice regularly.

When the fracture is complete and the symptoms more severe, two alternative forms of treatment are in common use.

Usually the foot and ankle are immobilized in a walking plaster extending from the metatarsal heads to just below the knee. The patient is taught to

walk in plaster and to do toe exercises. Immobilization is continued until pain and local tenderness have disappeared and radiographs show adequate union of the fracture. The usual period necessary is between six and eight weeks from the onset of symptoms. After the plaster has been removed, a course of Faradic foot baths and foot and ankle exercises help to restore full function.

A second method is advocated by those who consider the results of immobilization in plaster not wholly satisfactory. The foot is not immobilized, although a crepe or elastic bandage may be applied. The patient is either confined to bed or allowed to get about on crutches, but weight-bearing is not permitted. Regular foot and toe exercises are practised and the regime is continued until symptoms disappear and union appears to be complete.

Prognosis

A stress fracture of a metatarsal is usually regarded as a fairly trivial injury and indeed the results of treatment of these lesions in civilian life are almost universally good. In the Armed Forces, however, a 'March' fracture has acquired an evil reputation and a very high proportion of the personnel affected never return to full strenuous duties again. In athletes the same is true to a lesser extent; these fractures are often associated with a surprisingly long period of disability, and any attempt to resume full activity too soon is followed by a sharp recurrence of symptoms and disability.

STRESS FRACTURES OF THE OS CALCIS

The Os Calcis is a fairly common site for stress fractures, it has been suggested that perhaps 20 per cent of all these injuries occur in this bone.

Clinical findings

There is usually a history of marching, often on hard ground, after which the patient develops pain in the region of the heel. This pain is almost invariably gradual in onset, is relieved by rest and made worse by walking or marching.

On examination it is usual to find swelling in the region of the larger upper sub-achilles bursa, the hollows on each side of the achilles tendon are obliterated and the swelling may spread around the malleoli. Tenderness over the postero-superior aspect of the Os Calcis, that is over the fracture site, is always present.

Radiographs show the fracture line, which is in the vertical plane in the posterior part of the Os Calcis behind the subastragaloid joint. X-ray changes tend to be late, often appearing a week or two after the onset of symptoms.

The fracture line is usually incomplete, subperiosteal new bone formation on the upper surface of the Os Calcis is present and displacement is uncommon but not unknown. Simultaneous stress fractures of both Os Calces are not excessively rare.

Treatment

It is not often necessary to immobilize this fracture in plaster, avoidance of weight bearing by the use of crutches and instruction in foot exercises being the measures usually employed. The patient uses crutches until there is clinical and radiological evidence of union, which is to be expected in about eight weeks. Normal weight bearing and activity are then resumed gradually.

Prognosis

The prognosis after a stress fracture of the Os Calcis appears to be good. The fracture line does not involve the subastragaloid joint, there is no record of any late subastragaloid arthritis and recovery is complete.

STRESS FRACTURES OF THE FIBULA

Stress fractures of the shaft of the fibula are quite common and are interesting in that quite different fractures seem to be specifically associated with different activities.

Site

There appear to be two different types of stress fractures of the fibular shaft.

In the first type the fracture line is transverse and occurs in the lower third of the bone about two inches above the malleolus. This injury often follows running on hard ground and is said to be caused by the type of muscle contraction associated with running on the toes. An almost exactly similar injury occurs in skaters. Transverse fractures of the lower third of the shaft at a slightly lower level are seen in elderly patients who do not engage in any strenuous pursuit.

The second type is an oblique fracture of the upper third of the fibular shaft. This has been reported in service recruits undergoing some special training which involves jumping, and might be said to be a 'jumper's' as opposed to a 'runner's' fracture involving the lower third of the bone.

Clinical findings

A history of some special activity often provides a clue to the nature of the injury. This is followed by the development of pain of the usual type, that is, aggravated by exertion and relieved by rest, referred to the fracture site.

Tenderness over the fracture line is a constant finding and there may be some local swelling.

The fracture is shown by radiographs, and the bone changes seem to be early, being present in most patients when symptoms first appear. The fracture is usually complete but displacement is rare.

Treatment

The fibula is not a weight-bearing bone and the ankle joint is not involved by a stress fracture. Complete immobilization is, therefore, unnecessary and it is sufficient to cut down the patient's activity to a point at which symptoms disappear. When there is clinical and radiological evidence of union activity may be gradually resumed. When the patient is an athlete this resumption must be very carefully graduated indeed, if recurrence of symptoms is to be avoided.

Prognosis

Provided the period of rest is sufficiently long to allow complete consolidation of the fracture the prognosis is good and full function is regained. The immediate results are often disappointing in that symptoms tend to recur because strenuous pursuits have been resumed too soon.

STRESS FRACTURES OF THE TIBIA

Stress fractures of various types occur in the tibia, and although these varieties may overlap to some extent it seems clear that different activities produce typical forms of fracture.

Site

Complete transverse stress fractures occur both in the upper and lower thirds of the tibial shaft. When the upper third is involved the patient is usually young and engaged in strenuous activity, for example this type is commonly reported in recruits undergoing service training. The lower third fractures, on the other hand, are most often found in patients of middle age and in this group there is almost always a history of unusual exertion. These fractures are usually complete and some displacement is not uncommon.

Transverse fractures in the middle third of the shaft have been reported in ballet dancers sufficiently often to suggest that they may be associated with stresses peculiar to ballet dancing. Exactly similar fractures are, however, sometimes seen in middle-aged women of sedentary habit. When symptoms first occur these fractures are incomplete; but they may become complete or even displaced if dancing is continued.

Another type of incomplete fracture occurring about the junction of the middle and lower thirds of the bone is responsible for the so-called 'shin soreness' of athletes. These fractures are oblique and occur most often on the postero-medial aspect of the bone. Although the great majority of the reported examples of these fractures are incomplete it seems clear that if symptoms are ignored and running is continued they may become complete or even displaced.

Clinical findings

In the transverse and usually complete fractures of the upper and lower thirds of the shaft, the onset of symptoms is usually sudden and pain and disability is severe from the onset. As we have pointed out patients with upper third fractures are usually young while those with a lower third fracture tend to be of middle age. The clinical signs are tenderness at the fracture site, local swelling and bruising. These signs are much more obvious if displacement has occurred but so long as the fibula remains intact gross deformity does not occur.

Radiographs show the fracture which is not at this stage often associated with much evidence of repair.

The incomplete fractures, both of the transverse type in the middle third and the oblique type at the junction of the middle and lower thirds in athletes are much more insidious. At first discomfort occurs only during activity, usually running or dancing, is comparatively trivial and disappears entirely at rest. Pain gradually becomes more severe until it interferes with activity, at which point medical advice is usually sought.

Examination at this stage shows only tenderness and slight subperiosteal thickening at the fracture site and these signs are not always very obvious.

Radiological changes appear rather late and at first the radiographs may seem to be normal. Later, linear disruption of the trabecular pattern, absorption at the fracture line and subperiosteal callus make their appearance. The oblique fracture in runners is not always easily detected, and localized views of high quality taken from various angles may be necessary to demonstrate the bone changes.

Treatment

The tibia is a weight bearing bone exposed to severe mechanical stresses. For this reason all complete fractures must be immobilized in a full length plaster extending from the metatarsal heads to the upper third of the thigh. If the fracture is not displaced, weight bearing is permitted from the first. If displacement has been present crutches and no weight bearing is advisable for six weeks or so. The patient is instructed in toe and quadriceps exercises and static muscle contraction, and immobilization is continued until there is clear clinical and radiological evidence of union, which may be expected in between twelve and sixteen weeks. When union is satisfactory, the plaster is removed and the patient resumes activity gradually. Graduated foot, ankle and knee exercises are carried out regularly under supervision.

The incomplete fractures in which there is radiological evidence of repair are simpler to treat. Symptoms are usually controlled by stopping the exciting activity, and immobilization in plaster is not necessary. When local tenderness has disappeared and the radiographs show evidence of obliteration of the fracture line activity can gradually be speeded up until the patient has resumed a normal life. The trouble is that both ballet dancers and runners are usually anxious to get back to these pursuits as quickly as possible and it may be difficult to convince them they cannot with safety do this until bone healing is unequivocally complete.

STRESS FRACTURE OF THE TIBIA AND FIBULA

Stress fractures involving both the tibia and fibula are rare but have been reported.

Site

These fractures are usually transverse, both bones being involved at the same level. Both the upper and lower thirds of the bones may be the site of these injuries.

Clinical findings

Symptoms occur suddenly and are severe from the onset. Local tenderness and swelling are the cardinal signs and radiographs show the fractures at the onset of symptoms. Displacement at the fracture line is not uncommon.

Treatment

If they are displaced these fractures must be reduced and they must then be immobilized in plaster until union is sound. They unite rather slowly and

between four and six months immobilization is often necessary. The usual exercises are carried out in plaster, and, later, graduated exercises and a controlled return to activity are necessary.

STRESS FRACTURE OF THE PATELLA

Quite recently stress fractures of the patella have been reported in runners and it is probable that the course of this type of injury is such that it occurs from time to time without its true nature being suspected.

Site

The fracture line is transverse and may occur at any level in the patella. At first there is no displacement, but unless the bone is immediately protected separation of the fragments is inevitable; and when this stage has been reached the true nature of the injury cannot be detected.

Clinical findings

Some strenuous and sustained activity is followed by pain in the region of the patella. This pain may be severe but is not usually incapacitating. There is tenderness over the bone and some swelling of the knee joint with discomfort at the extreme of flexion. Radiographs show a linear transverse fracture of the patella without displacement. If the bone is not protected at this stage, displacement at the fracture line occurs within the next few days and the fragments become separated. The symptoms then become severe with incapacitating pain, a marked effusion into the knee joint and limitation of knee movement. At this stage the signs are obvious, and the gap between the fragments can usually be detected on palpation. Radiographs show the displaced patellar fragments, but there is nothing to suggest that this has occurred as the result of a stress rather than any other type of fracture.

Treatment

When this fracture is detected before displacement, the knee should be immobilized in extension in a plaster extending from the malleolus to the upper third of the thigh and kept in this position until the fracture has united. Weight bearing in plaster is permissible and static contraction of the quadriceps should be practised regularly. When there is clinical or radiological evidence of union, the plaster is discarded and very carefully graduated flexion exercises of the knee joint commence. Complete recovery is to be expected.

If separation at the fracture has occurred the management is the same as

that of any displaced fracture of a patella. Operation is necessary and either one or both of the fragments are excised and the quadriceps repaired or the two fragments are sutured together. The knee is fixed in extension in plaster until healing is complete and subsequently supervised graduated flexion and quadriceps exercises and a careful return to activity are necessary.

STRESS FRACTURE OF THE FEMUR

The femur is involved in stress fractures less often than the tibia or fibula.

Site

Stress fractures of the femur appear to be confined to two fairly well localized regions. Transverse fractures may occur in the lower third of the shaft, and the other area which may be affected is the femoral neck.

Clinical findings

Both fractures are usually associated with strenuous exertion but not with any particular form of activity. Both are insidious in onset and may be difficult to diagnose when symptoms first occur.

When the fracture involves the lower third of the shaft, the patient complains of pain in this region made worse by exertion and relieved by rest. The only finding is local tenderness, but the bone is deeply buried in the muscle and not easily palpable. Radiographs are frequently normal when symptoms first appear, and it may be a week or two before the fracture line and subperiosteal callus can be detected. Before the appearance of the X-ray changes it is impossible to diagnose this fracture with certainty. The clinical syndrome is sufficiently characteristic for a presumptive diagnosis to be made, and radiographs are then repeated at regular intervals until the appearance of the typical bone changes makes the nature of the condition clear.

Stress fractures of the femoral neck are also insidious in onset, and the initial symptoms are of pain in the region of the hip and a slight limp. On examination the joint is slightly irritable, with pain on and slight limitation of hip movement. At this stage radiographs are frequently normal. The firm diagnosis cannot be made until the typical fracture line appears; all that can be said is that the stress fracture is one of a number of possibilities. Radiographs should be repeated at regular intervals. When changes appear the fracture line may be complete or incomplete and, occasionally, if symptoms are ignored and the patient continues to engage in strenuous activity, dis-

placement may occur. If this happens the findings suddenly become those of a displaced fracture of the femoral neck with shortening and deformity of the leg, severe pain, limitation of hip movement and complete incapacity. The diagnosis is then obvious.

Treatment

Fractures of the lower third of the femoral shaft are rarely displaced and immobilization of the leg is not necessary. Rest in bed until the acute symptoms disappear should be followed by the use of crutches, weight bearing being avoided until the fracture has united. During the early stages of treatment active exercises should be practised with great discretion in order to avoid putting too much strain on the fracture line. Gentle static contractions of the quadriceps are followed by graduated knee exercises. Later, weight bearing is introduced gradually and activity is speeded up slowly, until the patient has resumed a normal life.

An undisplaced fracture of the femoral neck may be treated in one of two ways. The neck of the femur is subject to very severe mechanical strains both as the result of weight bearing and of movement of the hip joint. Displacement at the fracture line may be followed by serious late complications. For this reason the fractured femoral neck must be carefully protected until union is complete.

One method is to confine the patient to bed until symptoms disappear and then to permit the use of crutches, avoiding weight bearing until the fracture is united. This may take some time, it is usually three or four months before the radiographs show evidence of union. Weight bearing with normal activities should then be resumed very slowly but strenuous activity must be avoided for some months. During treatment on these lines active exercises must only be performed carefully and strenuous exercises must be avoided completely.

When the fracture is complete, with an obvious fracture line, some surgeons employ a different line of treatment. They consider that with conservative measures the danger of displacement is too great, and, even if this does not occur, the period of disability is unnecessarily long. For this reason they advocate immediate fixation of the fracture with a trifin nail. Following this operation symptoms quickly disappear and most surgeons permit modified weight bearing a few weeks later. Provided the patient leads a reasonably sedentary life there is no danger of displacement and union will take place slowly after this operation. When the fracture line has disappeared completely normal activities may be resumed.

If displacement at the fracture line has occurred immediate reduction of

the fracture and fixation with a trifin nail become urgently necessary. Treatment is then continued on the lines already indicated.

STRESS FRACTURES OF THE PUBIS

From time to time stress fractures of the pubis are reported. These are associated with some strenuous and often unaccustomed activity and are said to be due to repeated contractions of the adductor muscles.

Site

Stress fractures occur in the medial part of the pelvic ring. They are frequently incomplete.

Clinical findings

These fractures are associated with pain in the region of the pubis. This develops gradually and is aggravated by exertion, particularly by any exertion involving use of the adductor muscles. On examination there is tenderness in the region of the pubis and often pain in stretching the adductors.

The fracture, which is frequently incomplete and usually associated with new bone formation, is visible in the radiographs.

Treatment

The pubis is not a weight bearing bone and immobilization is not necessary. Strenuous activity is avoided, particularly anything which involves contraction of the adductor muscles. When clinical signs disappear activity may be resumed slowly, but unless strenuous exertions are avoided for two or three months a recurrence of symptoms is possible.

STRESS FRACTURES OF THE ULNA

In contrast to the leg, stress or fatigue fractures of the bones of the arm are extremely uncommon. This is probably accounted for by the different nature of the mechanical forces to which the arm is subjected. Stress fractures do occur in the ulna from time to time and appear to be almost invariably associated with lifting heavy weights with some form of implement.

Site

The mid-shaft of the ulna is the site of the injury and the fracture line is transverse.

Clinical findings

The patient gives a history of lifting heavy weights with a shovel or fork. This is followed by the development of pain in the forearm which comes on gradually and later becomes severe and incapacitating. On examination, there is swelling and tenderness over the fracture site, with some local swelling of the forearm.

Radiographs show a fracture of the typical fatigue type involving the shaft of the ulna. New bone formation is usually present.

Treatment

The arm is immobilized in plaster from the metacarpal heads to the axilla and immobilization is continued until union is sound. When the plaster has been removed a course of graduated exercises is necessary to restore full function.

STRESS FRACTURES OF THE HUMERUS

From time to time it has been stated that stress fracture of the shaft of the humerus occurs occasionally in athletes, discus and javelin throwers being particularly mentioned. The author has never seen such a fracture nor has he been able to find detailed records of one. From the theoretical point of view it seems unlikely that any 'whipping' action of the humeral shaft is likely to occur when an object is 'bowled' but might be produced by a 'throwing' action. The essential difference between these two movements lies in the use of the elbow joint. When bowling, the arm is held straight, that is fully extended at the elbow joint, and impetus is supplied by a shoulder swing aided by a body action of combined flexion and rotation. In throwing the elbow is held in flexion at the beginning of the movement, and impetus is added to the shoulder and body action by sudden extension of this joint at an appropriate moment. This sudden extension might be expected to cause whipping of the humeral shaft and might be responsible for a stress fracture. If this does occur, however, it must be an extremely rare phenomenon. If present trends continue it may be that such a fracture will be seen in the fast bowlers of the future.

STRESS FRACTURES OF THE FIRST RIB

Stress fractures of the first rib have been reported in athletes and in service men undertaking strenuous training.

Site

The most common site appears to be the anterior half of the rib and the fracture is transverse.

Clinical findings

Pain or discomfort in the region of the affected rib made worse by exertion is the usual complaint. On examination tenderness over the rib and a palpable swelling of this bone can be detected.

Radiographs show a fracture of the typical fatigue type, callus formation around the fracture line being present. Because of its position, and because it is obscured by surrounding bones, the first rib is not easily demonstrated radiologically and ordinary fractures of this bone sometimes occur as the result of muscular contraction without any very dramatic history of sudden symptoms. The differential diagnosis between a fatigue and an ordinary fracture is, therefore, a matter of some difficulty.

Treatment

Rest until symptoms have disappeared, followed by a gradual return to activity, are the only measures necessary.

Section III Spinal Injuries in Athletes

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1

STRUCTURE OF THE SPINAL COLUMN

THE spinal column is probably both the most important and the most complex structure in the entire locomotor system. Essentially it has three main functions, each of which demands quite different mechanical properties. Firstly it must supply stability to the whole body; it is the central core or strut to which the limbs, the bony thorax, the head and the main muscles of the trunk are attached and as such is subjected to enormous mechanical forces. Obviously a massive, rigid, bony structure would provide the greatest possible strength but any such arrangement would be incompatible with the second function of the spine, movement. The spine must be flexible, it must move freely in every direction under powerful muscle control, and this movement is a vital part of the mechanics of action. Finally, the spinal canal is the channel through which the spinal cord descends from the brain stem and in which the spinal nerves lie before leaving the canal at different levels to supply the various body structures. These nerves and, in particular, the spinal cord itself are extremely delicate structures and must be protected both from compression and stretching.

Any structure which fulfils these requisites, strength, stability, flexibility and the provision of a protective canal must necessarily be complex. Moreover the spinal column has been further complicated by the demands of evolution. When in late ologocene times man came down from the trees to walk upright on the ground he made novel demands on his spine and it is doubtful if the process of adaptation is yet complete.

THE STRUCTURE OF THE SPINE

The spinal column is made up of some thirty-three vertebrae articulating with or attached to each other. Various developmental abnormalities of distribution do occur but usually there are seven vertebrae in the cervical region,

twelve in the dorsal region, five in the lumbar region, five fused together making up the solid sacrum, and five rather rudimentary bones in the coccyx.

The bony vertebrae. Although the shape and size of the vetebrae vary greatly in the different regions and no two are exactly alike, their basic appearance is the same throughout the spine.

Essentially they consist of a solid body anteriorly with a posterior attachment, the neural arch. The bodies, the main weight bearing and force transmitting parts of the bone, are biconcave and roughly circular or oval in shape. (*Figure* 1).

The neural arches consist of two pedicles arising from the upper posterolateral aspects of the vertebral body and running backwards. These pedicles are strong and round in section and incline a little away from the mid-line. They terminate by joining the laminae, flattened plates of bone which incline towards the mid-line and are much deeper than the pedicles, their flat surfaces being tilted forward in the vertical plane. Posteriorly the laminae meet and fuse to complete the neural arch or ring.

From the arch seven different processes arise. At the junction of the pedicles and laminae the transverse processes project lateralward, being of varying size and strength in the different regions. From the upper surface of the pedicles at their laminar junction the superior articular processes arise and project upwards, their articular facets facing backwards. The inferior articular processes spring from the under surface of the laminae at their junction

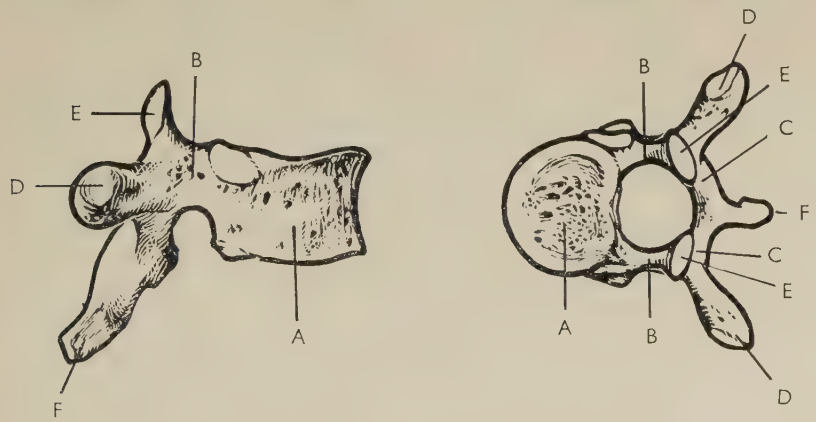

FIG. 1. *The structure of the bony vertebrae.* The vertebrae are basically the same in all regions of the spine, consisting of a solid body in front with a posterior attachment, the neural arch: (A = body, B = pedicles, C = laminae, D = transverse processes, E = articular processes, F = spinous processes).

with the pedicle and run downwards, their articular processes facing forwards. The spinous process arises from the posterior junction of the laminae and run backwards and downwards. (*Figure* 1).

The basic structure is common to all vertebrae but the size and shape vary widely in the different regions and even in these regions no two vertebrae are exactly the same, although they show the same general characteristics.

The intervertebral discs. Posteriorly the articular surfaces of the articular processes are in direct contact, forming synovial joints, but anteriorly the vertebral bodies are separated by the intervertebral discs. These discs are most complex structures and their mechanical functions are unique among the soft tissues of the body. The discs share with articular cartilage the distinction of being the only soft tissue habitually concerned in the transmission of mechanical force. At the same time they bind the vertebral bodies together, so that they can withstand huge mechanical forces without being torn apart; they permit movement between contiguous vertebrae; they help to control this movement and provide a fulcrum on which it can take place; they have a shock absorbing action; and by their size and shape they determine the total length and shape of the spinal column as a whole.

The adult disc consists of three parts, the cartilage plates enclosing it above and below, the annulus fibrosus and the nucleus pulposus.

The cartilage plates cover the central part of the upper and lower surfaces of the vertebral body. The fibres of the annulus and nucleus take origin from and are intimately blended with the peripheral part of this cartilage plate.

The annulus fibrosus consists of a series of concentric lamellae whose fibres run obliquely from their attachment to the cartilage plates and marginal rings of adjacent vertebral bodies. The fibres of the different lamellae lie at an angle to each other, an arrangement which increases the total strength of the annulus. The number of layers varies in the different regions of the spine but they tend to be packed closer together anteriorly and posteriorly and thicker and more widely spaced on the lateral sides of the discs. Anteriorly and posteriorly, the outer fibres of the annulus blend with the powerful anterior and posterior common ligaments and through these gain attachments to the anterior and posterior surfaces of the vertebral bodies. The anterior ligamentous attachment is, however, much stronger than the posterior and the posterior ligament is relatively a much weaker structure than the anterior. The annulus fibrosus binds the vertebral bodies together but, because of its lamellated structure and the direction of its fibres, permits a certain amount of movement between the individual vertebrae. The annulus also serves as a retaining envelope for the nucleus by which its fibres are slightly distended.

The adult nucleus pulposus is composed of a loose network of fine strands

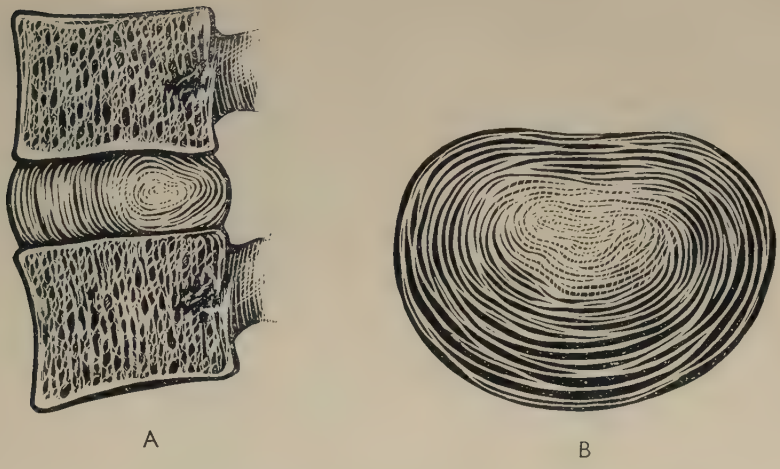

FIG. 2. *The intervertebral discs.* These lie between the vertebral bodies: they consist of an outer layer of concentric fibrous lamellae, the annulus, which encloses a central network of fibrous tissue containing semi-gelatinous material, the nucleus.

of fibrous tissue containing semi-gelatinous material. The nucleus as a whole is situated rather behind the centre of the disc, is slightly plastic, obeys the law of fluids and is held in shape by the surrounding annulus and cartilage plates. (*Figure* 2 A and B).

There is no clear cut line of demarcation between annulus and nucleus; they merge one into the other and, with increasing age, the difference between them becomes progressively less distinct. The nucleus constitutes rather less than one-quarter of the total volume of the disc. The function and mechanics of the intervertebral discs are discussed more fully in later sections.

The overall shape of the spine. The spinal column as a whole is not straight but curved and there are four distinct curves in the normal spine. The primary curves are concave forward and correspond to the curve in the embryonic axis. These primary curves, which occur in the dorsal and sacral regions, are caused by the shape of the vertebral bodies themselves, the dorsal and sacral vertebrae being deeper posteriorly than anteriorly. The secondary curves are convex forwards to permit the assumption of the erect position and are largely due to the shape of the intervertebral discs. These secondary curves occur in the cervical and lumbar regions. In the neck the secondary curve begins to develop during late foetal life and is produced by the shape of the cervical discs, which are deeper anteriorly than posteriorly. The secondary lumbar

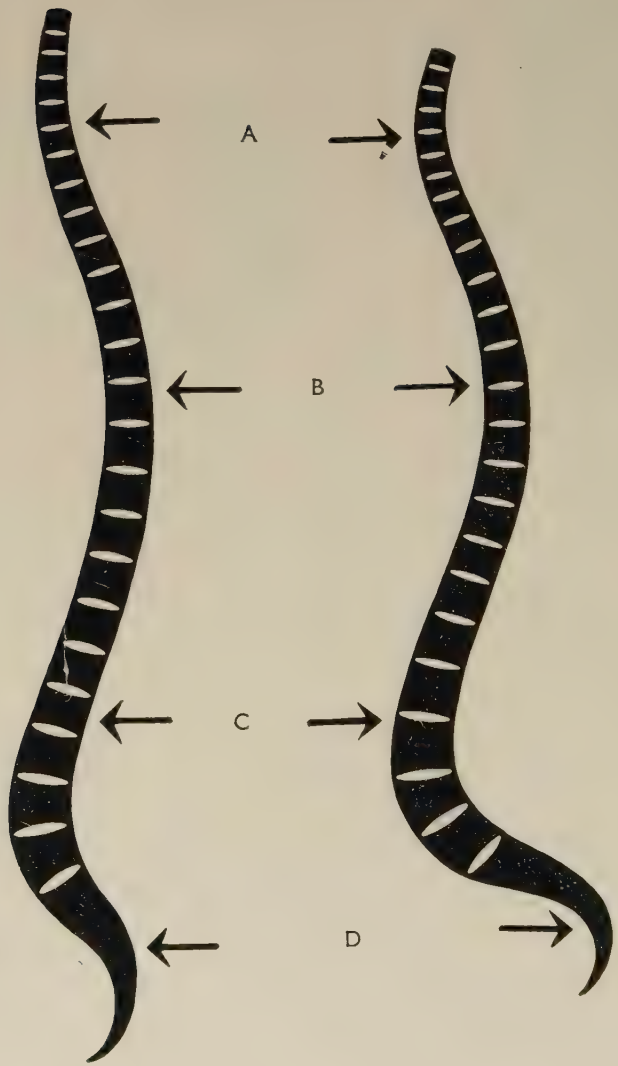

FIG. 3. *The overall shape of the spine.* There are four distinct curves in the spinal column. The primary curves (B and D) are concave forwards and lie in the dorsal and sacral regions. The secondary curves (A and C) are convex forwards and lie in the cervical and lumbar regions. Normally the curves are more accentuated in the female than in the male.

curve develops after birth with the extension of the legs and later with the assumption of the sitting and standing position and is largely due to the shape of the lumbar discs. In the lower races the total depth of the anterior surfaces of the vertebral bodies is just less than that of the posterior surface and, in such races, the lumbar curve is due entirely to an increased anterior depth of the discs. In the higher races the anterior depth of the bodies is just greater than the posterior depth but the major part of the anterior convexity is produced by the shape of the discs.

At the lumbo-sacral junction there is an acute angle in the spine. This varies from individual to individual and is greater in the female than the male. It first appears about mid-foetal life and is entirely due to the shape of the L_5/S_1 disc which is, on an average, some two-and-a-half times deeper anteriorly than posteriorly. (*Figure* 3).

While the pattern of curves in the spinal column is constant in all normal individuals the amount of curve varies to some extent from person to person and is greater in the female than in the male. Too great an increase in curvature in the spinal column leads to various mechanical disorders which will be discussed later.

The total length of the spinal column varies greatly from individual to individual, between one-quarter and one-fifth of the overall length is due to the intervertebral discs and these are relatively thicker in the more mobile regions of the spine, that is in the neck and lumbar regions. There is a slight diurnal variation in the thickness of the discs themselves produced by the fluid exchange between the discs and the adjacent tissues.

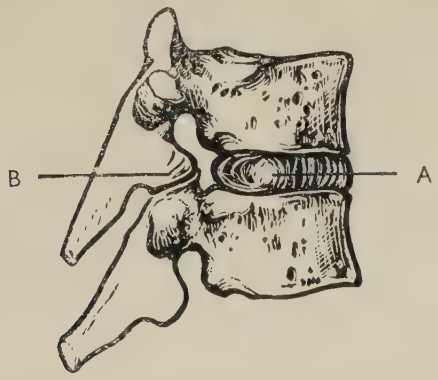

FIG. 4. *The intervertebral articulations.* The intervertebral joint consists of three parts; the anterior joint between the vertebral bodies (A) and two posterior joints between the articular processes (B).

The intervertebral articulations. The intervertebral joint between contiguous vertebrae consists of three parts, an anterior amphiarthrodeal joint between the vertebral bodies and two posterior diarthrodeal joints between the articular processes. The intervertebral disc is an integral part of the anterior joint and permits a small range of movement between the vertebral bodies. The posterior joints are diarthrodeal with articular cartilage, synovial membrane and a joint capsule. The plane of these joints in relation to the long axis of the spine varies in different regions and determines the plane of movement possible at that level. Both anterior and posterior joints are reinforced by powerful ligaments and protected by the spinal muscles. (*Figure* 4).

The mechanics and the range of movement of these joints is discussed in a later section.

The spinal ligaments. The spinal column is reinforced by a series of powerful ligaments which bind the vertebrae together and check spinal movements.

The anterior longitudinal ligament is a strong fibrous band extending from the occiput above to the front of the sacrum below and lying on the anterior surfaces of the vertebral bodies. It consists of several fibrous layers, the most superficial extending over three or four vertebrae and the deeper passing from one vertebra to the next. The fibres are firmly fixed to the intervertebral discs and to the margins of the vertebral bodies and are less firmly attached to the central part of the anterior surfaces of the vertebral bodies.

The posterior longitudinal ligament lies on the posterior surface of the column within the spinal canal. It is attached to the discs and to the margins of the vertebral bodies and it varies in shape at different levels. In the cervical region it is of uniform width while in the dorsal and lumbar regions it is denticulated, being narrow over the bodies and wider over the posterior surfaces of the discs. The posterior longitudinal ligament is not as powerful a structure as the anterior ligament.

The neural arches are connected by a series of ligaments. The ligamenta flava connect the laminae of adjacent vertebrae, being attached to the lower part of the anterior surface of the laminae above and the posterior surface and upper margin of the laminae below. These ligaments extend from the capsule of the posterior articulations in front to the mid-line behind, where their posterior margins are in contact and blend to some extent. The interspinous ligaments connect adjoining spinous processes and extend from the ligamenta flava in front to the supraspinous ligament behind. The ligamentum nuchae is a fibrous membrane in the neck corresponding to the supraspinous ligament of the thorax and lumbar region. Above it is attached to the protuberance and crest of the occiput, and it runs downwards to the spine of the seventh cervical vertebra, being attached anteriorly to the spines of all the

cervical vertebrae and forming a septum between the muscle groups on each side of the neck. The supraspinous ligament is a strong cord of fibrous tissue connecting the tips of the spinous processes from the seventh cervical vertebra to the sacrum. As it descends this ligament becomes thicker and stronger. The intertransverse ligaments connect certain transverse processes. In the cervical and lumbar regions these ligaments are relatively flimsy structures but they are stronger in the dorsal region. (*Figure* 5).

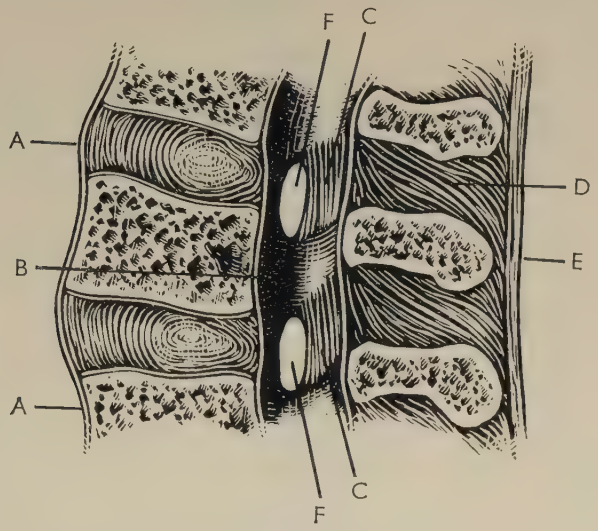

FIG. 5. *The spinal ligaments.* The spinal column is reinforced by powerful ligaments. The anterior and posterior longitudinal ligaments (A and B) join the vertebral bodies and intervertebral discs, while the ligamenta flava, the interspinous ligaments and the supraspinous ligament join the neural arches (C, D and E).

The spinal canal. The spinal canal may be described as a flexible and protective tube through which the spinal cord descends from the brain stem. The bony constituents of the canal provide the protective element while the ligaments joining these permit movement between them and render the canal as a whole flexible. On cross section the canal is roughly circular, but its exact shape varies in the different regions of the spine and indeed to some extent from person to person. Its overall size in an adult also varies with the bone structure of the individual.

The anterior wall of the canal is formed by the vertebral bodies and the posterior surface of the intervertebral discs, covered by the posterior longi-

FIG. 6. *The spinal canal.* The spinal canal lies within the neural arches and extends from the foramen magnum in the skull above to the sacrum below (A). The spinal nerves emerge from the canal through the spinal foramina (B).

tudinal ligament. The lateral walls are formed by the ligamenta flava, the posterior intervertebral joints and the pedicles. Below each pedicle and in front of the posterior intervertebral joints lie the intervertebral foramina. Posteriorly the canal is roofed by the ligamenta flava and the laminae. Because of their obliquity, only the upper third of the anterior surfaces of the laminae are in immediate relationship to the canal, the rest of the posterior wall consisting of the ligamenta flava. In the middle line these ligaments meet and fuse with each other to some extent, the junctions being covered by the overlying interspinous ligaments. (*Figure* 6).

The foramina are the apertures through which the lumbar roots leave the spinal canal. Their upper and lower boundaries are formed by the vertebral pedicles, the anterior walls are formed by the postero-lateral aspect of the vertebral bodies and the intervertebral discs and the posterior boundaries are the articular processes forming the posterior intervertebral joints. The free lateral edge of each ligamentum flavum forms part of the posterior boundary. (*Figure* 6 B and 5 F).

The major part of the canal is occupied by the meninges and their contents; the cerebrospinal fluid, the spinal cord above and the nerves of the cauda equina below. In the adult the spinal cord ends at the level of the first lumbar vertebra and, below this, the nerves of the cauda equina pass downward inside the theca for some distance before they traverse it to leave the canal. The course of the nerve roots after they leave the spinal cord is important. After leaving the cord they run downwards within the theca for varying distances, the lower the root the greater being its intrathecal course. They then pierce the anterior wall of the theca and continue downwards within the canal for some segments before leaving through the intervertebral foramina.

The spinal theca is in contact with the walls of the canal everywhere and is surrounded by a varying amount of fat and loose areolar tissue. In this tissue lie the spinal arteries and veins and the spinal nerves in their extrathecal course.

The muscles of the spine. The spinal column as a whole has an adequate range of movement in all directions although the plane and nature of this movement varies greatly from region to region. The importance of spinal movement will be discussed in a later section but it is, of course, under powerful muscular control.

The arrangement of the muscles and muscle groups controlling spinal movement is extremely complex. It is, however, possible to differentiate between three main types of muscle action in relation to the spine; postural, prime movers and secondary movers.

Many muscles are concerned with posture, that is they maintain the re-

lationship between the individual vertebra and 'brace' the spine to withstand force. Of these the most important are the complex group of deep extensor muscles of the spine. This group, extending from the sacrum to the occiput, produce both extension, lateral flexion and, to some extent, rotation of the column. The group has, in fact, a double function. It is one of the keys to good body posture, maintaining as it does the erect position. It is also a prime mover of the spine, producing extension either from the flexed or neutral position. The spine is also 'braced' and posture maintained by other muscle groups. In the lumbar region the spine is supported by the psoas, one of the long flexors of the hip and further supported anteriorly by pressure generated by the abdominal muscles. The dorsal spine is supported anteriorly by the muscles of the thorax and the muscles of respiration while in the neck the cervical muscles all help to maintain posture in relation to the head.

The second type of muscle action is that contributed by those groups which may be described as 'prime movers' of the spine. The function of the deep spinal extensor muscles in this relationship has already been discussed. Flexion is produced by the abdominal and cervical muscles, and lateral flexion by the oblique abdominal muscles, part of the deep extensor muscle mass and by unilateral action of the flexor and extensor muscles. Rotation is produced by parts of the extensor muscle mass and by the cervical muscles.

The third group of muscles acting on the spine may be described as 'secondary movers'. All the muscles connecting the arms to the trunk and legs to the pelvis, the muscles joining the skull to the trunk and the muscles of respiration are primarily concerned with movement of the arms, legs and head or with breathing but all have secondary effects on the spine in that they produce or tend to produce spinal movement.

It is clear, therefore, that a very large number of the muscles of the body have either a direct or potential action on the spine. The position becomes even more complex during activity and particularly during strenuous activity when the appropriate spinal movement is necessary to produce maximum efficiency in almost any type of physical action.

2

THE SPINE IN MOVEMENT AND POSTURE

THE MOST superficial observation shows that quite apart from the differences associated with age, sex, race and size, individuals vary widely in appearance and physical characteristics. Although not so immediately obvious there is an equally wide variation in temperament and mental ability.

Far less attention is paid, however, to a third individual characteristic, posture and manner of movement. These differ from person to person almost as much as facial appearance or personality, but only in the extreme forms do they attract any attention. We may perhaps speak of some one as being clumsy or ungainly, and of another as being graceful or lithe, when these characteristics are outstanding, but beyond this very little notice is taken of how we stand or move. Yet a good posture and a proper manner of moving are, in a large measure, the key to general physical well-being and certainly their possessor avoids many conditions which are common sources of discomfort and ill-health. Moreover, these are not necessarily or even often inborn characteristics, but are in most cases acquired by physical culture in some form. Their acquisition is not difficult nor does it demand any unusual physical attribute, a good carriage and the ability to move well is within the reach of anyone who is prepared to take a little trouble.

The most important single factor in a correct posture and fluent movement is the spinal column, its role in both is vital.

SPINAL MOVEMENTS

In the spinal column stability and flexibility are combined in a most effective way and for such a strong and massive structure the range of movement normally present is surprisingly large. The various regions are not equally mobile, however, nor is movement in all the different planes uniformly distributed over the whole length of the spine.

Spinal movement can be considered from two aspects, the movements between contiguous vertebrae and the sum of these movements, that is the movement of the column as a whole.

FIG. 1. The axis of movement of the spine as a whole passes through the posterior half of the vertebral bodies, traversing the nucleus of each intervertebral disc.

Movements between individual vertebrae

In the pre-sacral spine some degree of movement is possible between each individual vertebra and the next. The range and plane of this movement varies greatly but the mechanism is basically the same in each region.

The joints between the vertebrae are compound, each consisting of three parts. The anterior part between the vertebral bodies is formed by the intervertebral disc. This structure, unique among the body tissues, permits the vertebral bodies a limited but universal range of movement in relation to each other. Posteriorly there are two diarthrodial joints between the upper and lower articular processes of the vertebrae concerned. These joints permit a limited range of movement but the plane of their articular facets determines its direction.

The intervertebral joint is a very complex mechanism, depending for its proper functioning on the integrity of all its separate parts, and of these the intervertebral disc is by far the most important. In any type of joint the two most important mechanical points are the fulcrum on which and the axis around which movement takes place. Normally the articular surfaces of the bones provide a fulcrum, and the axis of movement is determined by the shape of these surfaces. In the case of the intervertebral joints, however, the only direct contact between the vertebrae is in the posterior articulations. The articular processes are not sufficiently strong to provide a fulcrum for movement of a joint subjected to enormous forces, and the axis of movement and the fulcrum lie well in front of the posterior joints.

In fact, the axis of movement in the spinal column passes through the posterior half of the vertebral bodies, parallel to the spinal canal and traversing the nucleus of each intervertebral disc (*Figure* 1). This axis is probably not absolute, but shifts forwards and backwards a little in different phases of spinal movement. The fulcrum for movement between the individual vertebrae is provided by the nucleus pulposus. As has been pointed out, the nucleus obeys the law of fluids, that is to say it is incompressible and therefore provides a suitable fulcrum. Its shape is not fixed but is determined by the action of the slightly stretched elastic fibres of the annulus fibrosis by which it is contained. The shape of the nucleus alters a little according to whether the spine is in the extended, neutral or flexed position. (*Figure* 2). This slight alteration in the shape of the nucleus accounts for the shift in the axis of movement of the intervertebral joints. Movement between each individual vertebra is, of course, under muscle control, and the range of movement is limited by the fibres of the different layers of the annulus and the intervertebral ligaments becoming taut and, in certain regions of the spine, by bony contact between different parts of the vertebrae themselves. The spinal

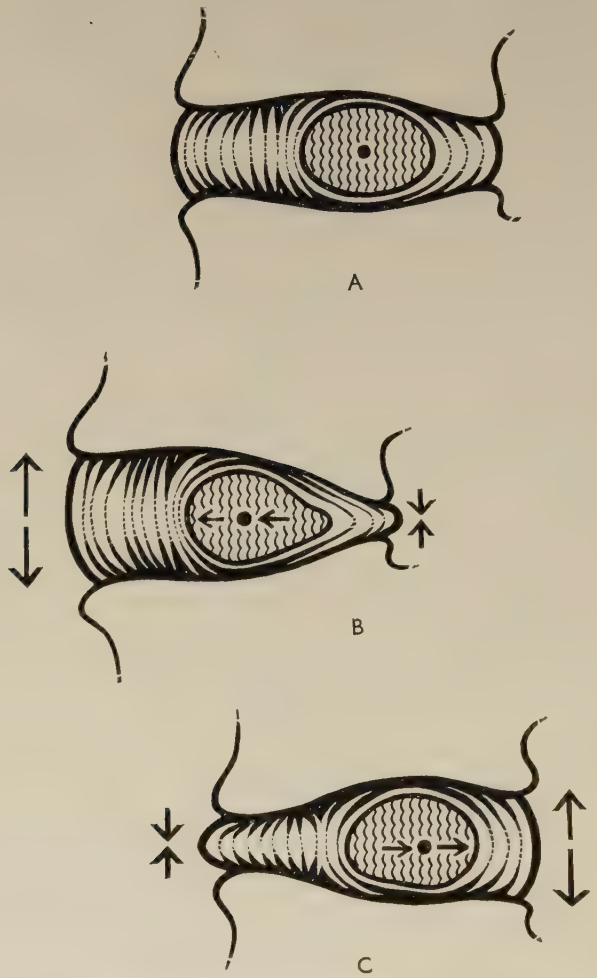

FIG. 2. The incompressible nucleus supplies a fulcrum for movement between individual vertebrae (A). The shape and position of the nucleus alters slightly on movement; there is a little forward shift on extension (B) and a backward shift on flexion (C).

muscles also control the range of movement. The plane of the articular surfaces of the posterior joints has a most important relation to rotatory movement of the spine. In the lumbar and cervical regions these surfaces lie across the plane of rotation around a central axis, so that this movement is impos-

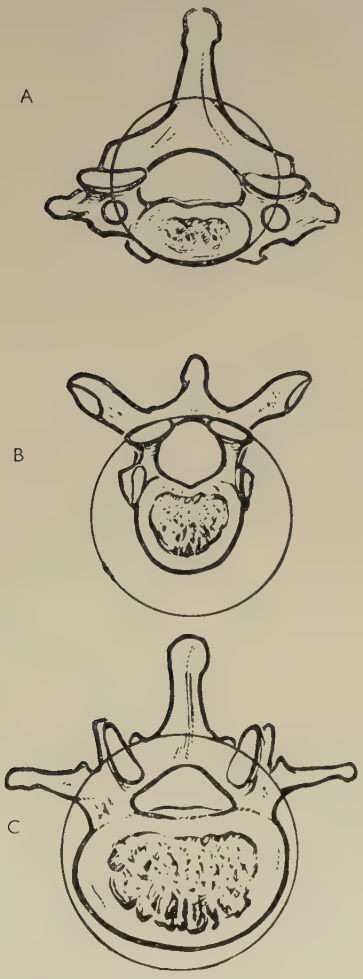

FIG. 3. Rotation of the spine is controlled by the plane of the articular surfaces of the posterior intervertebral joints. In the cervical (A) and lumbar (C) regions, these lie across the plane of rotation around a central axis, preventing this movement. In the dorsal region (B) on the other hand, the articular facets lie in the plane of rotation, allowing this movement to take place freely.

sible, while in the dorsal region on the other hand these surfaces lie in the plane, so that the dorsal vertebrae can rotate freely in relation to each other. (*Figure* 3).

The mechanical forces to which each intervertebral joint is subjected are very great and the nature of these forces is discussed later.

Movements of the spine as a whole

It is impossible to divorce movements of the spine from body movement as a whole. Indeed the ordinary methods of examining the range of spinal movement in fact measure the flexibility of the whole body rather than that of the spine. Provided that this is recognized, however, these methods are of great value. The normal range of spinal or, more accurately, body flexibility, is determined by a series of fairly simple observations. The subject stands erect with the feet together and the knees straight and, from this position, moves in all directions, the range of each movement being noted.

Forward flexion is usually measured by the distance between the finger tips and the ground at the extremes of this movement. Normal young adults can just about touch their toes but some can place their hands flat on the ground without difficulty while others can only reach their ankles. Extension is observed when the subject bends backwards as far as possible, although there is no simple method of measuring this movement. Lateral flexion can be measured by the distance between the extended fingers and the ground at the extremes of this movement and rotation by the number of degrees through which the head and shoulders can be turned, the feet remaining still.

It must be emphasized, however, that these measurements are only approximate; wide variations occur in perfectly normal individuals and other joints, notably the hips, are concerned in these movements.

The most mobile regions of the spine itself are those in which the intervertebral discs are thickest, the lumbar and cervical. Apart from the range of movements, however, the planes in which movement is possible differ in different areas.

In the neck the first important joint is the compound articulation between the occiput, the atlas and the axis, which is found between the head and the neck. The upper two cervical vertebrae, the axis and the atlas, are highly modified for this purpose. The outstanding feature of these two vertebrae is the solid bony process which projects upward from the body of the axis and in fact represents part of the body of the atlas which has become separated from this bone. This permits a wide range of rotation but prevents flexion and extension between these two bones. On the other hand the shape of the articular surfaces at the atlo-occipital level permits free flexion and extension between the skull and atlas but prevents rotation. The head, therefore, can be flexed and extended in relation to the fixed atlas and axis, while the head and

atlas rotate as one in relation to the fixed axis. In the remainder of the cervical region the movements of flexion, extension and lateral flexion take place, the range of extension is somewhat greater than that of flexion and lateral flexion is associated with a little rotatory movement. True rotation is, however, impossible, because of the plane of the articular facets.

In the dorsal region the only free movement is rotation. The plane of the articular facets, unlike those of the cervical and lumbar regions, lies in the axis of this movement. Flexion, extension and lateral flexion are extremely limited, particularly in the upper dorsal region, being prevented by impingement of the vertebrae on each other and by the bony thorax.

In the lumbar region the intervertebral discs are relatively thick. If the whole spine is considered the intervertebral discs account for only between one-quarter and one-fifth of the total length but in the lumbar region about one-third of the total length is made up by the lumbar discs. As one would expect, therefore, the lumbar region is the most mobile in the whole spine; and here flexion, extension and lateral flexion take place freely. The range of extension is greater than that of flexion or lateral flexion, and rotation is prevented completely by the plane of the posterior articular facets. It is difficult to compute the exact range of spinal movement accurately although there are, of course, many different ways in which this can be done. For example, radiographs can be taken of the spine at the extremes of movement, and by making tracings of the different bones the degree of tilting of these bones in relation to some fixed point can be calculated. Such methods are of limited practical value and it is sufficient to remember that while the spine is remarkably flexible considering its structure, it takes rather less part in the total range of body movement than one might expect. For example, when normal adults touch their toes, movement in the lumbar region, that is in the most flexible part of the spine, only accounts for about twenty per cent of the total forward bending and at the extremes of this movement the anterior convexity of the lumbar spine is only just obliterated.

Although the range of spinal movement is not great in relation to the range of body movement as a whole, movements of the spine play a vital part in the locomotor system.

Mechanical forces acting on the spine

The mechanical forces acting on the spine are both very large and extremely difficult to compute with any pretence at accuracy. Such forces arise from three sources—gravity, muscle tone and bodily activity.

While it is impossible to measure these forces with any form of instrument, some theoretical estimate of their potential may be arrived at. It is usual to

consider them at the point where they are greatest, that is in relation to the L5/S1 intervertebral disc, the fulcrum on which the lowest joint of the mobile part of the spinal column moves. At this level in the erect position, the force due to gravity is the weight of the upper part of the body, that is thorax, arms, head and neck. To this must be added any weight carried in the arms or supported on the shoulders or head. The second source of force is the tone of those muscles attached on either side of the L5/S1 intervertebral joint, that is the erector spinae muscle mass and the abdominal muscles. The role of muscle tone in 'bracing' the spine, and the immense importance of this, is discussed later in relation to posture and movement. There can be no doubt that contraction of these muscle groups can produce very considerable forces of a compressing nature. The degree of such force certainly varies very widely, when the muscles are completely relaxed it is probably quite small, being then due only to the inherent elasticity of the muscles themselves. When the muscles are fully contracted, however, so that the body is 'braced' to withstand some shock or perform some strenuous action, this force rises steeply and may very well reach heights much greater than we think. Finally, during the course of normal life the L5/S1 joint, like all the other intervertebral joints, is exposed to multiple and rapidly varying forces associated with ordinary activity. These are made more complex by the fact that they are magnified by the lever action of the trunk and limbs. In the ordinary course of events the greatest forces are probably produced by creating the longest possible lever, that is by forward flexion of the trunk and extension of the arms. If for example a weight of 100 lbs. were lifted in this manner, it must be balanced by contraction of the extensor muscles of the spine acting much closer to the fulcrum of joint movement, that is to the intervertebral disc. The relation between the long anterior and short posterior levers has been computed as 15 to 1; and the 100 lbs. must, therefore, be balanced by muscle contraction of 1500 lbs., the total force falling on the joint in these circumstances being 3000 lbs., about one-third of a ton. (*Figure* 4). In a similar way all body actions produce forces falling on the spine, these forces being magnified by levers created by the limbs and trunk, and all such forces must be counter balanced by contraction of the appropriate muscle group acting through other levers of varying lengths.

It is, therefore, clear that the spinal column is subjected to mechanical forces which are not only enormous but which are extremely and rapidly variable. These forces are greatest at the lower limit of the mobile spine, that is the L5/S1 intervertebral joint, and decrease upwards, being least in the cervical region.

FIG. 4. Ordinary activity subjects the spinal joints to multiple and rapidly varying forces which are magnified by the levers created by the body itself. For example, forward flexion of the trunk and extension of the arms creates a long anterior lever, and forces acting on the joints through this lever must be balanced by muscle contraction acting through a much shorter posterior lever. The relation between these levers has been computed as 15 to 1; thus a weight of 100 lbs lifted in this way must be balanced by muscle contraction of 1,500 lbs., throwing a total force of 3,000 lbs. on the intervertebral joint.

The spine and body movement

The mechanics underlying even the simplest movement are complex. It is not just a matter of the contraction of a muscle so that its origin and insertion are brought nearer, with consequent movement of any joint over which it passes. Associated with contraction of the prime movers or activating muscles there is a synchronized relaxation of the appropriate antagonists, and also contraction of other muscles which stabilize joints which, while not directly concerned in the primary movement, might be affected by the muscle action producing it.

Further, even the simplest movement is rarely confined to one part, and the more strenuous and complex the activity the more widely is the body involved. Indeed, the whole locomotor system takes part in most of the ordinary everyday activities, for example, when we walk movements of one limb are counter-balanced by or associated with movements of the other limbs, the trunk and the head and neck. The correlation and blending of all these separate actions produces a balanced fluid motion which cannot be reproduced by any mechanical contrivance. The synchronization and effectiveness of

movement varies widely from individual to individual and many animals move much better than man; but it is only necessary to compare any normal adult in motion with a puppet activated by strings or a mechanical toy to realize how rich is our physical endowment. The ability to move well is not inborn, however; during childhood, as our bodies develop, we learn to use, co-ordinate and control our muscles and to some extent this ability is lost in old age. Two factors determine any single individual's abilities, his inborn physical characteristics and the amount of physical education he receives and of these the latter is by far the most important. It is a little strange that education is always interpreted as a training of the mind, and as such is a matter of great concern to a large number of people, including our legislators. On the other hand, physical education, the training of the body, which is equally, if not more, important to our well-being is largely left to the individual.

The spinal column, as the central core of the body, has a vital part to play in motion. Overall balance of the body in motion is largely maintained by spinal movement, the effectiveness of action of any single limb is largely dependent on spinal movement which places the body in the most advantageous position for the task in hand, and the muscles of the spinal column are a source of very great power which is utilized in many activities. During activity, therefore, the spine is in constant motion under powerful muscle control. The range of its movement is not necessarily great, but without it much of the physical effectiveness of the body as a whole is lost.

The proper use of the spine is not a simple matter and can only be acquired by training. Good posture is not a static phenomenon to be considered only in relation to sitting or standing, on the contrary, posture during activity is of vital importance. The spinal column is the key to good posture either at rest or during activity.

The spine in posture

To appreciate the importance of posture, either moving or static, it is necessary to have a clear understanding of the effects of the transmission of force on the tissues and structures of the body. All body tissues are constantly subject to varying stresses, either compressing or stretching. These stresses are intermittent and of different magnitude, but they all tend to distort the tissue on which they act. Rigid structures, such as bone or cartilage, can resist considerable strains: soft structures are easily compressed or stretched. Even soft structures, however, can sustain some amount of force without damage, and some fibrous structures, for example joint ligaments, are so

strong that they can tolerate considerable stretching forces without damage.

The effect of a force on body tissues depends on two factors, its magnitude and its duration. The influence of the first factor is comparatively simple, if the stress is greater than the strength of the structure to which it is applied injury occurs, a bone is broken, soft tissues are torn or stretched, a blood vessel may be ruptured and so on. Such occurrences are responsible for all accidental injuries. The second factor, the duration of application of force, has a much more subtle influence. With one exception all body tissues are harmed by the habitual, that is to say, the constant long-maintained transmission of force. For example, a relatively small stress falling on a ligament may be easily sustained and cause no damage if it is of short duration or intermittent; but if it is constant over long periods, it sets off a cycle of pathological changes, loosely classified as 'strains' which result in disfunction and disability. These changes are not dependent on the magnitude of the stress but on its duration, and they are responsible for a very large number of disorders. The one exception to this rule is bone, which can withstand compressing force indefinitely without damage; indeed bone tends to become stronger in proportion to the forces which it has to bear. The habitual transmission of stress is, therefore, one of the main functions of the skeletal system. Since the skeleton must be articulated to allow movement, two other types of body tissues are inevitably involved in the habitual transmission of force. These are articular cartilage and the intervertebral discs. Both are highly modified for this function and, in the main, carry it out efficiently but, unlike bone, neither becomes stronger by so doing, and both are subject to a number of 'wear and tear' disorders. These factors are the key to the role of the spine and, indeed, the rest of the skeleton, in good posture, both active and static. The essential feature of such a posture is first that the body should be so disposed that all the main stresses are transmitted by bone and secondly that it should be 'alerted', that is in a position from which movement in any direction can be initiated easily.

As far as the spine is concerned, the forces to which it is subjected, gravity, muscle tone and muscle action, have already been considered. These forces are transmitted along the column and ultimately reach the ground through the legs or are expended in action. While they are being transmitted by the spine it is essential that they should pass through the vertebral bodies and intervertebral discs and not fall on the posterior joints, the spinal ligaments or the spinal muscles. The factor which determines the line of transmission over the spine is, of course, the relation of the vertebral bodies to each other. As has been pointed out, when a man stands erect in the natural or mid-position the spine is not straight but is convex forward in the lumbar and cervical regions

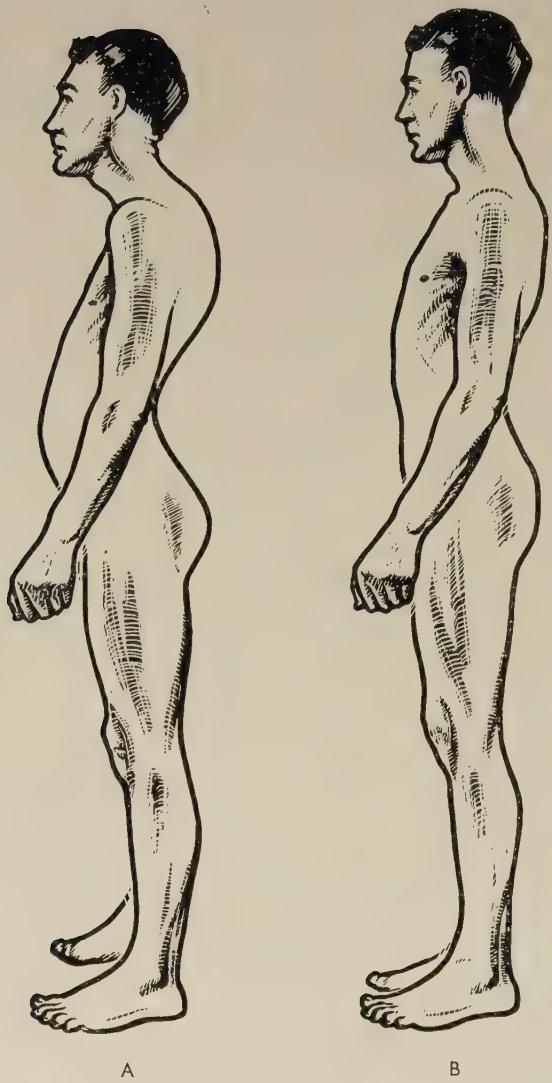

FIG. 5. A poor posture with undue accentuation of the normal curves of the spine causes forces to fall on the soft tissues (A). When the posture is good, these forces pass through the vertebral bodies (B).

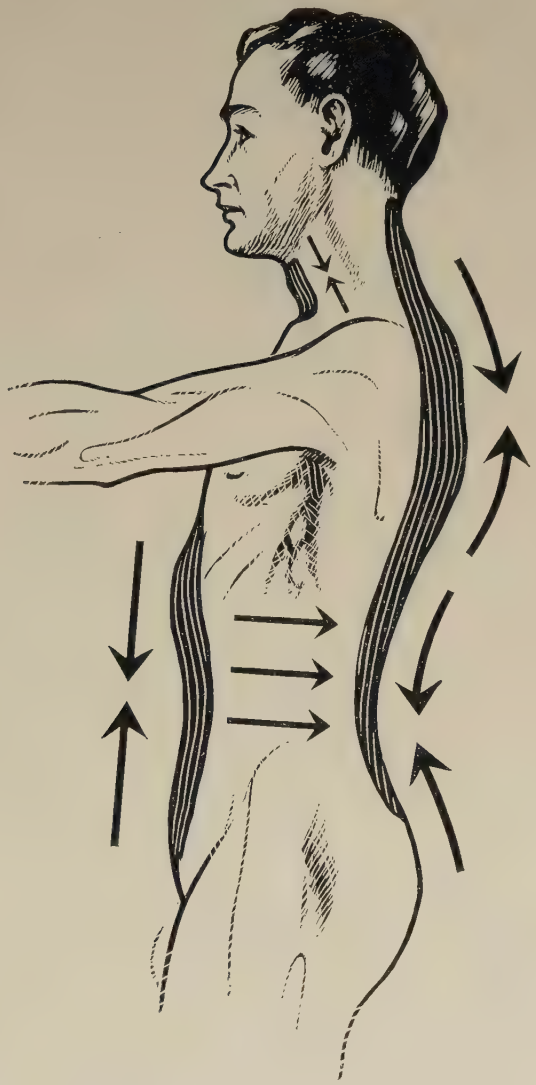

FIG. 6. The forces primarily responsible for a good posture are the muscle tone of the erector spinae mass behind and the abdominal muscles in front. The abdominal muscles act by producing a positive intra-abdominal pressure which falls on the anterior aspect of the lumbar region.

and concave forward in the dorsal region. The degree of curvature varies from individual to individual and is greater in the female than the male; but, provided it is not unduly accentuated, the main line of force transmission passes through the vertebral bodies and intervertebral discs. If, for any reason, the normal curves are unduly increased, however, forces fall on the soft tissues. If these forces are too great damage may occur, while if they are too long maintained the cycle of chronic stress changes is started. (*Figure* 5).

It is therefore essential that the spine be maintained at all times in such a position that the stresses to which it is subjected are transmitted by the vertebral bodies and the discs if injury or chronic changes are to be avoided. This position can only be maintained by the muscles controlling the spine, the most important being the erector spinae mass behind and the abdominal muscles, acting through intra-abdominal pressure, in front. (*Figure* 6). Unless spinal posture is good the posture of the body as a whole either at rest or moving cannot be other than bad.

The development of spinal posture

Good spinal posture is so important in the prevention of injury and of chronic stress disorders associated with the habitual transmission of force through soft tissue, that no pains should be spared in its acquisition. Two factors are involved in the development of such posture, one physical and one mental. From the physical point of view the essential feature is to build up and strengthen the muscle groups acting on the spine, the erector spinae and the abdominal muscles being the most important. This is done by a graduated series of muscle building exercises; and it is vital to remember that these are aimed at strengthening the muscle groups and not at increasing the range of spinal mobility, which can be a dangerous pursuit, involving as it does the stretching of the ligaments which limit these movements. In other words it is better to have an average range of spinal movement under very strong muscle control than a wide range of movement associated with less powerful control. The second factor, the mental outlook, is of equal importance. A conscious effort to maintain good spinal posture at all times and to resist any tendency to 'slump' on to one's ligaments must be maintained until the habit is so established that it slips under the level of consciousness. Even when this stage has been reached, from time to time a mental reassessment of one's physical attributes and, in particular, of one's posture and manner of movement is an essential and often a salutary exercise.

3

TRAUMATIC LESIONS OF THE SPINE

THE STRENGTH of the spinal column is so great that very considerable violence is necessary to damage it. Indeed the only really common disorder of a mechanical nature is a lumber or cervical disc lesion and the role of trauma in the production of these lesions is highly controversial. Because major damage to the spine only occurs in 'high velocity' accidents or in those in which huge mechanical forces are involved and because minor damage is relatively uncommon, spinal injuries are not frequent occurrences in the course of games and sport.

SOFT TISSUE INJURIES OF THE SPINE

Fractures or fracture-dislocations of the spine are always associated with extensive injury to the soft tissues, that is to the muscles and ligaments in the vicinity. Indeed this soft tissue damage is often so severe and widespread as to over-shadow, both in its immediate and late effects, the actual bone or joint injury. Trauma in various forms may, however, affect soft tissues alone without any involvement of either the bones or joints. The structures affected are the spinal ligaments, the muscles and, possibly, the fascial layers. These injuries take the form of a stretching or tearing of muscle or fibrous tissue and are produced either by stretching associated with abnormal body movement or by violent muscle contraction.

Injuries to the spinal muscles

The muscles directly associated with the spinal column are, in the main, of the short fleshy variety, the range of spinal movement is less than that of the large joints in the limbs and the column itself is relatively stable. Because of these factors, the spinal muscles are less liable to injury than those in the arms and legs. Stretching or tearing of these muscles unassociated with any other injury does occur from time to time in sport. These injuries are rarely produced by an abnormally large range of movement. If such a movement, however produced, is sufficiently extensive to tear muscle fibres it also almost always either tears one of the spinal ligaments, disrupts the intervertebral joints or fractures the vertebrae themselves. Isolated muscle tears usually

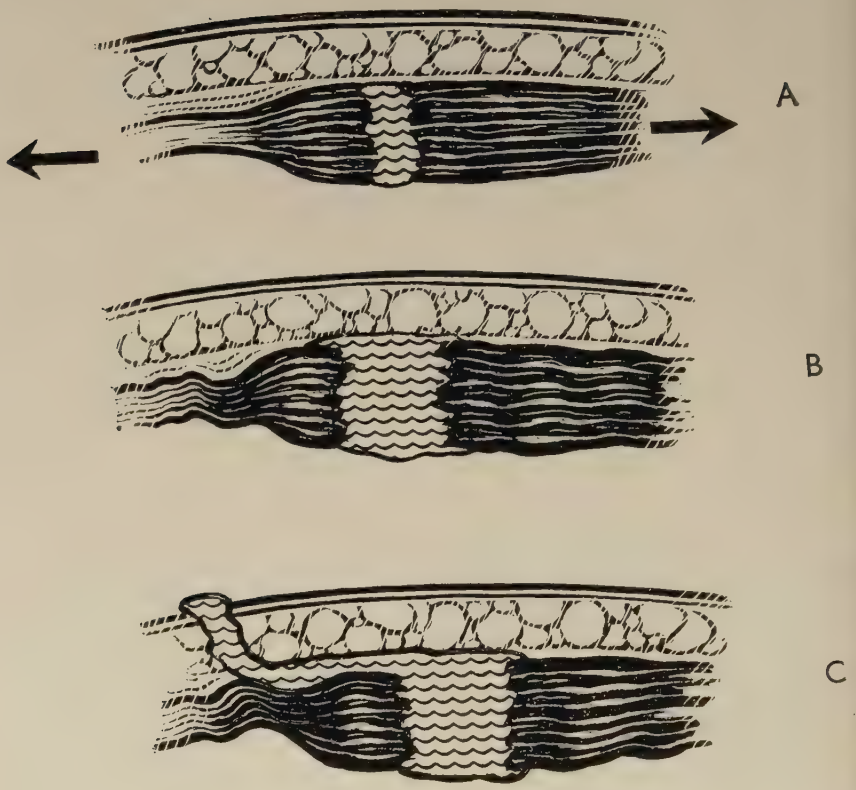

FIG. 1. When a muscle is torn, the space between the ends of the ruptured fibres fill with blood (A). If the muscle is completely divided, the ends retract leaving a large gap which is filled with blood (B). This blood ultimately tracks to the surface, producing a haematoma which may appear some distance from the site of the muscle injury (C).

occur as the result of abnormal muscle contraction, often associated with some unbalanced or involuntary convulsive movement.

The commonest site of such tears is the erector spinae muscle mass. Here the damage is usually confined to a fairly small area of muscle, often involving a few fibres only. The injury is usually at or close to the origin or the insertion of the muscle, or at a point where fleshy muscle fibres join a tendon or tendinous sheath.

The symptoms of muscle damage are a sudden sharp pain at the point of injury followed by localized pain whenever the damaged muscle is actively

contracted or stretched by movement. On examination, local tenderness can be elicited and this is followed by local swelling. Some internal bleeding at the site of the injury always occurs but the extravasated blood may track along the fascial planes and only become subcutaneous and visible as bruising some distance from the site of injury. If the tear is sufficiently large, an actual gap may be felt on palpation of the muscle; and the fleshy fibres above or below the tear contract and may be felt in the form of a soft tumour. (*Figure* 1). A further diagnostic test is the complete disappearance of symptoms when the area is infiltrated with local anaesthetic. This manoeuvre abolishes all pain arising from the area anaesthetised but does not affect bone or joint injuries, should these be associated with the muscle damage.

The treatment of these muscle tears is based on the same principles as those applied to muscle injuries elsewhere. Natural healing involves organization of the haematoma which is ultimately replaced by fibrous tissue, bridging the gap and uniting the ends of the torn muscle fibres. Obviously, the larger the gap and the more extensive the damage, the longer this process takes. More-

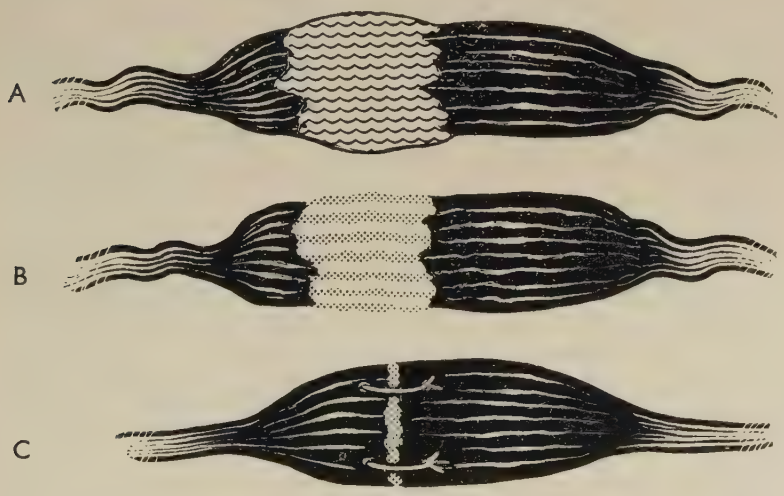

FIG. 2. When a muscle is completely torn, the ends retract, leaving a gap which becomes filled with blood clot (A). If healing then takes place, this blood clot is replaced with fibrous tissue and the continuity of the muscle is restored. The muscle is, however, lengthened and its function thereby impaired (B). If the gap is obliterated by early surgical repair, that is, by suturing the torn muscle ends together, healing is more rapid and the muscle is restored to its original length with no loss of function (C).

over even when healing is complete the muscle fibres are not joined together, the gap is bridged but not obliterated so that the muscle is lengthened and its function may consequently be impaired (*Figure* 2).

If, therefore, early surgical repair of the damaged muscle is practicable, the time necessary for healing is reduced and normal function of the damaged muscle is preserved. Exposure and suture of muscle tears can only be satisfactorily carried out if the tear is fairly large and the damaged muscle reasonably accessible; and this is rarely the case when the spinal muscles are involved.

Failing surgical repair, the only treatment is rest until the damage heals naturally. While healing is taking place any active contraction or passive stretching of the affected muscle tends to tear the newly formed tissue and may delay or prevent healing. Rest is provided by avoidance of activity, possibly aided by some form of splinting. It is doubtful if the process of healing can be materially speeded up by any other measures. The injection of various substances into the affected area has always been popular, possibly because of its dramatic appeal. If local anaesthetic is used symptoms are temporarily abolished, and this measure is often employed as a short term method of overcoming disability. The danger is that further damage may occur while pain, which is both a natural warning and a basic part of the protective mechanism of the body, is artificially abolished. The substances most popular today are Cortisone preparations of various kinds. There is no theoretical justification whatever for the use of these substances in such a manner, their local action delays natural healing and may even, with repeated use, prevent it altogether.

Muscle tears, even small tears, are often surprisingly persistent in their effects, and may cause disability out of all proportion to their mechanical significance. This is because the fibrous tissue scar formed in the process of healing is often sensitive to stretching and causes pain whenever the muscle is vigorously contracted. At this stage, that is when the healing is complete, local measures calculated to soften and stretch the scar and strengthen the affected muscle are often of value.

Damage to the spinal ligaments

Most of the ligaments of the spine are associated with the intervertebral joints and for this reason any ligamentous damage usually constitutes a joint strain or sprain. These injuries are discussed in a later section.

There is one group of spinal ligaments, however, which may be damaged without associated joint injury; those between the spinous processes. Such injuries take the form of stretching of part or all of the ligament or actual

tearing of the ligamentous fibres, the extent of the area involved varying between a few fibres only and complete rupture of the whole ligament.

Damage to the interspinous and supraspinous ligaments. These ligaments become taut when the spine is flexed, and an abnormal range of flexion may damage them in the lumbar and lumbo-dorsal regions, producing a condition which has been called 'sprung back'. Such injuries may occur in the course of various sports.

The clinical picture is of a sudden localized pain on flexion, followed by local tenderness and bruising. Flexion of the spine causes a recurrence of local pain and this movement is limited by associated spasm of the erector spinae muscle mass. The symptoms are abolished by infiltration of the area with local anaesthetic.

Treatment consists of the avoidance of flexion of and flexion strains on the spine and the wearing of a supporting corset until the damaged ligament has healed. At the same time extension exercises are carried out regularly to prevent loss of tone or wasting of the extensor muscles.

'STRAINS' OR 'SPRAINS' OF THE SPINAL JOINTS

Much of the confused thought so prevalent in regard to spinal disorders stems from the terminology in common use in both medical and lay circles. Not only are many of the terms applied to common conditions ill-conceived, misleading, or even frankly incorrect, but confusion is made worse because they are employed in a thoroughly haphazard manner.

The term 'strain' or 'sprain' of a joint is one of those most frequently misused. These terms, which are not quite synonymous, can only be properly employed when applied to a joint which has been subjected to a mechanical stress sufficient to stretch or tear part of the capsule or joint ligaments but not sufficient to cause dislocation or subluxation of the joint as a whole. A 'strain' implies a simple stretching of capsular or ligamentous fibres and is usually produced by a small stress applied over a long period while a 'sprain' means that some of these fibres are actually torn, usually by a greater but transitory force. In almost all sprains the synovial membrane of the joint is also damaged. Both these injuries are followed by other changes in and around the affected joint, but the essential feature of both conditions is that they are of traumatic origin, involving primarily the joint capsule or ligaments. One might imagine that this was clear beyond the possibility of any confusion, but such is not the case. There are various fairly common disorders in which changes in the spinal joints, either intra-articular or peri-articular, are associ-

ated with chronic changes in the joint capsule or ligaments. These changes are purely secondary in nature and the primary condition is usually easily recognized, yet the term 'strain' or 'sprain' is often freely and quite erroneously applied to all such conditions.

All the joints of the spine are, of course, constantly exposed to injuries, both major and minor, and these may occur in the course of various sports. In their major forms such accidents produce fractures or fracture-dislocations, but gross violence is necessary to disrupt the spinal column in this way. Lesser forces may damage the spinal joints and produce true 'strains' or 'sprains'. On the other hand the spine, as has been pointed out, is strong and these joints are protected by powerful muscle groups, so that these injuries are much less common than might be expected.

There are three types of 'strain' or 'sprain' affecting the spinal joints.

Sacro-iliac strain. 'Sacro-iliac strain' is probably second only to the kindred condition of lumbo-sacral strain in being the most abused diagnosis commonly applied to low back disorders. These two conditions form a sort of diagnostic scrap heap to which are assigned all patients with low back pain of obscure origin.

Although the sacro-iliac is a synovial joint, its main function is the transmission of weight and, architecturally, its design is calculated to provide a maximum amount of stability. The articular surfaces are irregular rather than flat, and the irregularities on the sacral and iliac articular facets interlock and restrict joint movement. This locking device permits the transmission of weight without the production of ligamentous tension. The sacro-iliac ligaments, in particular the anterior sacro-iliac ligament, are themselves very strong and only permit a very limited range of joint movement. Normally antero-posterior movement, appreciably greater in the female than in the male, is the only movement which takes place at this joint. Anatomically, therefore, the picture is of a powerful and stable joint at which only a very limited range of movement is normally possible, a joint which habitually is subjected to powerful stress and which would not appear likely to be affected by anything short of a very violent injury.

It is possible, however, that during the course of various games or athletic pursuits this joint may be subjected to force sufficient to produce a true strain or sprain, although it is a very rare occurrence. The patient suffering from a sacro-iliac strain or sprain gives a history of injury immediately followed by pain localized to the region of the joint. This pain persists in the form of a dull and localized ache, made worse by exertion and sharply exacerbated by rotatory movements of the pelvis. On examination, pain is felt in the affected joint and deep tenderness is present in the area. Some local discomfort usu-

ally occurs at the extremes of all movements of the spine, this being most marked on rotation or lateral flexion away from the site of the injury. Sharp local pain may be produced by straining the affected joint by rotating the pelvis with the patient supine.

Radiological examination shows no abnormality; subluxations of the joint of a degree sufficient to be visible only occur in the puerperium, in some paraplegic conditions and in association with some fractures of the pelvic ring.

It should be remembered that, in certain disc lesions, pain and local tenderness may be referred very accurately to the sacro-iliac region. This happens when the posterior primary division of one of the lower lumbar roots has borne the brunt of the intraspinal root involvement, and such patients are often wrongly considered to be suffering from sacro-iliac strain.

The treatment of sacro-iliac strain is rest in the acute phase, exercise to improve the extensor muscles of the spine and the general posture and, in certain circumstances, a supporting corset.

Lumbo-sacral strain. Reference has already been made to the common misuse of this term. Some authors even go so far as to list various disorders of the joint as 'causes' of lumbo-sacral strains. Such an attitude indicates a complete failure to grasp the basic pathology either of these conditions or of joint strain.

The lumbo-sacral joint differs from the other intervertebral joints in several respects. With the exception of the sacro-coccygeal joint it is the lowest in the vertebral column and for this reason force transmitted between the spine and the pelvis falls directly on the lumbo-sacral joint without being 'buffered' in any way by being transmitted through a lower articulation. The sacro-iliac joint is situated at an acute angle in the vertebral column, this angulation being produced in part by the shape of the L5/S1 disc, and the transmission of force across an acute angle always presents difficulties. Finally, the plane of the posterior lumbo-sacral articular facets does not coincide with the plane of the other posterior articulations. These features might be expected to make the lumbo-sacral joint more susceptible to injury than the other intervertebral articulations.

The patient suffering from lumbo-sacral strain gives a history of injury followed by pain in the low back. The initial severe pain is replaced by dull aching, which becomes acute on any sudden or extreme movement of the joint. These symptoms are persistent, moderately severe and aggravated by exertion. On examination, symptoms are referred accurately to the joint, and a deep tenderness may be present in this region. There is a little spasm of the lower lumbar muscles and slight limitation of all movement of the lumbar spine with pain or discomfort at the extremes of these movements. Quite

sharp local pain is often produced by extending the spine from a fully flexed position.

Radiological examination shows no abnormality.

It should be remembered that a lesion of the L5/S1 disc itself produces a disorganization of the lumbo-sacral joint. The diagnosis of lumbo-sacral strain implies an injury to the joint in the form of a stretching or tearing of its supporting ligaments, but without associated damage to the intervertebral disc. Such an injury is probably quite uncommon and is certainly a much less common cause of chronic low back pain than a disc lesion at this level.

The treatment of lumbo-sacral strain is rest until the acute phase has passed, followed by a supporting corset, extension exercises to improve the posture and avoidance of excessive strain on the joint until the symptoms have disappeared completely.

Strains of the intervertebral joints. Dislocations, fracture-dislocations and subluxations of intervertebral joints occur as the result of injury, these lesions being most common in the cervical and lumbo-dorsal regions. They are produced by mechanical forces of a severe and localized nature. Lesser forces may produce a true sprain, that is capsular or ligamentous damage without any joint disorganization. Such injuries may occur from time to time but it is probable that, with the possible exception of the cervical region, they are very rare.

The clinical picture of an intervertebral strain or sprain is of an injury, usually in the form of an abnormal movement, followed by local pain. On examination the pain is found to be referred to the region of the injury and is associated with muscle spasm and limitation of movement of the affected joint. Pain is sharply aggravated by forcibly reproducing the movement which produced the injury. In the cervical region the initial symptoms are often severe and muscle spasm may produce an obvious postural deformity.

In a true strain or sprain the X-rays show no abnormality.

The treatment is rest in the initial stages, possibly reinforced by splinting of the affected area of the spine and, later, exercises to improve the posture and build up the spinal muscles so that they protect the damaged joint.

4

TRAUMATIC LESIONS OF THE SPINE (Continued)

FRACTURES OF THE SPINE

THE DIAGNOSIS of a 'broken back' or 'broken neck' has the most sinister connotation to the average layman. Such injuries are generally regarded as being rare, and either fatal or followed by severe and permanent disability. In fact, fractures of the spine are relatively common and the vast majority are neither serious nor likely to cause subsequent disfunction, provided they are adequately treated. Spinal fractures or even fracture-dislocations do occur in the course of various games and sports and it is, of course, important to recognize and treat them adequately.

Simple fractures of the spine fall into various classes, depending on the part of the vertebra involved.

1. *Fractures of the spinous processes*

Fractures of the vertebral spinous processes are uncommon. Apart from occasional freak injuries due to a direct blow on the centre of the back when the spine is in flexion, such fractures only occur as the result of muscular contraction. Such an injury occurs during the shovelling of heavy earth or clay, this particular action producing a violent contraction of the rhomboid muscle which may snap the spinous process of the seventh cervical or first dorsal vertebra.

The occasional fracture of a spinous process seen in athletes is usually due to a direct blow from a ball or other object when the spine is flexed. The symptoms are those of local pain or tenderness sharply aggravated by forward flexion. The presence of such a fracture can be confirmed by radiographs.

These injuries are not usually associated with prolonged discomfort or subsequent disability and the patient need only avoid flexion strains and do regular exercises for the extensor muscles.

Fractures of a spinous process rarely unite by bone; but the fibrous repair is sufficiently strong to prevent subsequent disfunction. In those occasional patients in whom symptoms persist the fractured fragment can be excised completely and the soft tissues repaired by suture.

2. *Fractures of the transverse processes*

Fractures of the transverse processes are confined to the lumbar region where they are produced by violent contraction of the quadratus lumborum muscle. Such a contraction usually occurs as the result of an accident in which the lumbar region is forced into lateral flexion, this movement being resisted by violent spasm of the quadratus lumborum muscle. One or more transverse processes are broken or avulsed and there is associated widespread tearing of muscle fibres.

Following this injury there is deep tenderness in the flank on the affected side. Sharp pain is produced by contraction of the torn muscle, that is by active flexion of the trunk towards the injured side, or when the injured muscle is stretched, that is by passive lateral flexion of the trunk away from the injured side. All other movements of the lumbar spine are associated with some discomfort in the flank.

Radiological examination of the lumbar region shows the fractures.

Although fracture of one or more transverse processes are in themselves relatively trivial the associated muscle tear may be the source of prolonged and severe disability. For this reason the treatment of these injuries is important. In the early stages a period of rest is necessary, and this is best achieved by rest in bed during which time gentle extension exercises of the spine are practised. When the acute symptoms have subsided activity is resumed gradually, and some surgeons prefer to immobilize the lumbar region in a plaster jacket at this stage. Gentle extension exercises are continued, and as soon as pain and muscle spasm have disappeared the plaster jacket is discarded and graduated exercises are started and increased slowly until full movement and power is restored.

3. *Fractures of the articular processes*

Fractures of the vertebral articular processes occur in conjunction with fracture dislocations of the spine and are discussed in a later section.

4. *Fractures of the vertebral bodies*

Fractures of the bodies of the vertebrae, either single or multiple, occur fairly commonly. These fractures are of various types, but all are produced by a compression force which crushes the vertebra.

The exact nature of the vertebral damage depends on the position of the spine when the compressing force is applied. If, as is most commonly the case, the spine is in flexion or forced into flexion as the compressing force is applied, the vertebral body is wedged anteriorly. (*Figure* 1). Such an injury is produced by a fall from a height when the spine is flexed on impact, by a

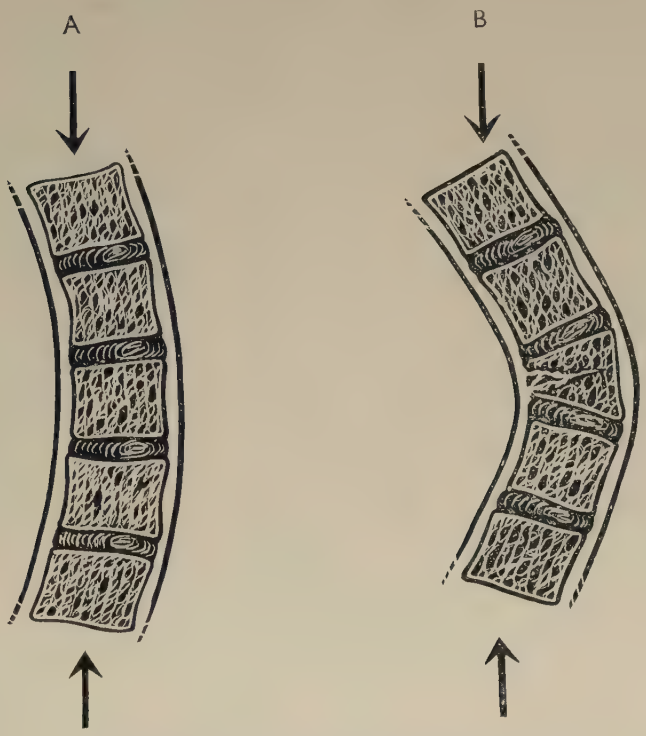

FIG. 1. If the spinal column is in flexion or is forced into flexion when a compression force is applied, the vertebral bodies may be crushed into a wedge shape, the wedge pointing anteriorly. This is the common type of vertebral body fracture.

weight falling on the shoulders when the spine is in flexion, and by similar types of injury. If the whole spine is subjected to the force, as in a fall, several vertebrae may be affected. Much the commonest site for such injuries is the lumbo-dorsal junction, the least common the lower cervical region. They may, however, occur at any point in the column if the flexion force is sufficiently well localized.

Occasionally the compression force is applied when the spine is in the neutral position. The vertebral body or bodies are then compressed much more uniformly: but, because of the influence of the posterior intervertebral joint, some anterior wedging tends to occur.

In rare cases the compression force is applied with the spine in extension, or the force itself hyperextends the spine. When this happens the vertebral

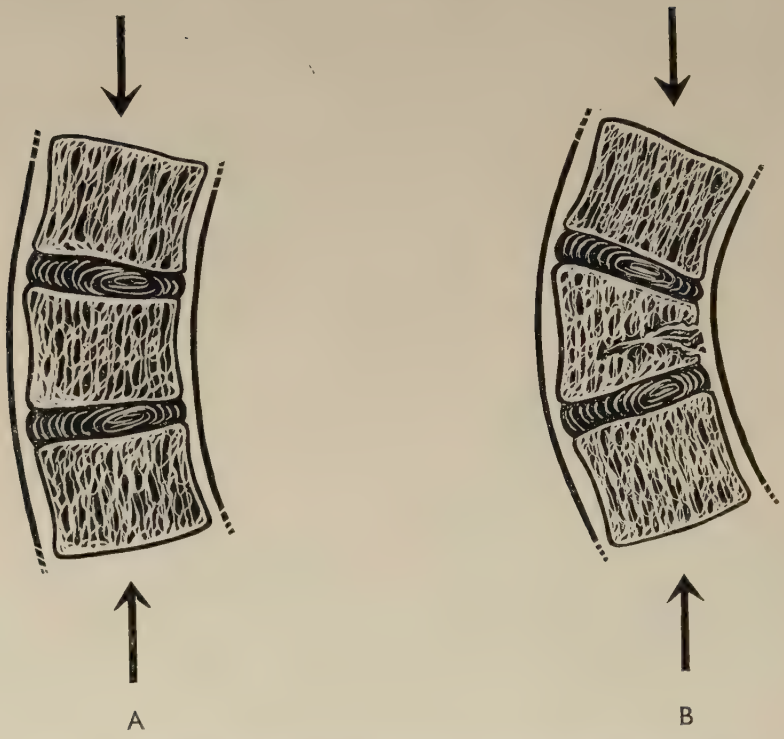

FIG. 2. Occasionally a compression force is applied to the spine when it is in extension, or the force itself extends the spine. If a vertebral body is fractured in these circumstances, it is compressed posteriorly and assumes the shape of a wedge pointing backwards.

body is compressed posteriorly, producing the so-called extension fracture. (*Figure* 2).

A fracture of one or more vertebral bodies is associated with local pain, muscle spasm and limitation of with pain on movement of the affected region. The clinical findings of local tenderness, muscle spasm and limitation of with pain on spinal movement and the history of the injury suggests a fracture of a vertebral body and the diagnosis is confirmed by radiography.

Treatment of vertebral body fractures is based on the principles which govern the treatment of any fracture. If gross anterior wedging is present an attempt may be made to reduce the deformity by extending the spine in various ways, and subsequently it is immobilized in a plaster jacket or a plaster collar until union is complete. During the period of immobilization

extension exercises should be carried out assiduously and when union is complete progressive spinal exercises are necessary to restore full mobility and power. Recently there has been an increasing tendency to consider accurate reduction of a wedge deformity of a vertebral body of less importance than was formerly supposed to be the case. There is good reason to believe that, provided the intervertebral articulations are not grossly damaged and the extensor muscles are powerfully built up, the shape of the body itself will not prevent the recovery of normal function.

Occasionally a fracture of a vertebral body is associated with permanent damage to an intervertebral joint above or below and is followed by persistent local pain, limitation of movement and disability. In these circumstances a local operative fusion of the spine may be necessary and, provided the whole of the damaged area is rendered solid, the results of operation are highly satisfactory.

DISLOCATIONS AND FRACTURE DISLOCATIONS OF THE SPINE

Dislocations and fracture-dislocations of the spine are more serious injuries than uncomplicated fractures. Not only are they more difficult to manage and more likely to cause subsequent disability but when the spinal column is itself disrupted the contents of the spinal canal, that is the spinal cord or the cauda equina or the spinal nerves in their intraspinal course, may be damaged with serious and often permanent consequences.

The effects of injury on the spinal cord and the spinal nerves are extremely important. The cord itself consists of various ascending and descending tracts, conveying sensations of different kinds from the body to the brain and messages from the brain to the different body structures. Within the cord are central columns of cells which are relay stations in these tracts. Broadly speaking the afferent or sensory tracts lie posteriorly and the efferent or motor tracts anteriorly in the cord. When the fibres making up these tracts are divided they are incapable of regeneration, that is the changes are permanent. In certain circumstances, however, conduction along these fibres may be interrupted temporarily without the fibres themselves being disrupted and in such injuries conductivity may return when the immediate effects have passed off. For example, such temporary effects follow a contusion of the cord with local oedema and conductivity subsequently returns as the oedema is absorbed.

Fracture-dislocations or dislocations of the spine are invariably associated with distortion of the spinal canal and as the result of this distortion the spinal cord is often stretched, crushed or even actually torn. If, as the result of such

injuries, the fibres of the tracts are divided, conductivity is then permanently abolished. If the distortion is not sufficiently great the cord may only be contused and, as has been pointed out, the disturbances of function may then only be temporary in their effects. When the conductivity of the cord is interrupted, either temporarily or permanently, the immediate effect is a complete flaccid paralysis of the body below the level of the injury. All feeling and power of movement is lost and the limbs are limp and useless. When the damage is permanent the nature of the paralysis later changes; the muscles become spastic and certain reflexes return, but there is no sensation and no power of movement. The damage may not affect the whole cord when the effects will depend on the site and extent of the damaged area. For example, sensation may be lost but the power of movement remain, or vice versa, or one half of the body may be involved but not the other. The effects of contusion are initially the same as those of section; but within two or three weeks sensation and movements start to return and, provided no fibres have been permanently damaged, recovery will ultimately become complete, although this may take many months.

Damage to the nerves in their intraspinal course differs from cord damage. Conductivity in these nerves may be temporarily interrupted by compression or stretching, or the nerve fibres may actually be divided. Provided the nerve remains in continuity, that is provided the nerve ends at the point of division are not actually separated, regeneration can take place. Nerve fibres grow distally within the nerve sheath until, ultimately, the whole nerve is re-established. As with the cord, nerve conductivity may be temporarily interrupted by contusion and subsequent oedema without the fibres being divided, and in this case the function returns as the effects of this injury disappear. When the conductivity of a nerve is interrupted its function is lost and, depending on its nature, sensory or motor changes or both are produced. After contusion these changes disappear within a few weeks but if the nerve fibres are divided regeneration is a very slow process, fibres grow distally at a uniform rate and the period necessary for recovery depends on the length of nerve to be replaced.

Dislocation or fracture-dislocations of the spine may involve both the cord and the intraspinal nerves at the level of the injury. Below the level of the L1/L2 intervertebral joint the cauda equina may be involved but the cord itself cannot be affected.

Sensation and movement are not the only functions disturbed by cord injuries. The fibres controlling the viscera are also affected and cord damage is associated with visceral disturbance of which bladder and rectal paralysis are the most important.

Fracture dislocations in the dorsal or lumbar spine

Fracture dislocations of the dorsal and lumbar spine occur mainly in the region of the lumbo-dorsal junction, although in special circumstances they may occur elsewhere. They are usually produced by a flexion force, often associated with a rotatory strain. If the force is applied over a short section of the spine the damage tends to be more severe, since it is not buffered or taken up by spinal movement as it might be when a longer segment is affected. The sort of accident associated with a lumbo-dorsal fracture-dislocation is a crush injury such as might occur when a miner, working in a low shaft in a flexed position, is trapped under a rock fall, a sudden flexion strain on impact after a fall from a height, or a high velocity localized flexion injury in a car or aircraft crash. Lumbo-dorsal fracture-dislocations of the spine are unusual, but by no means unknown, in sport.

The immediate effect of a fracture-dislocation of the spine is severe local pain sharply aggravated by any movement. If the spinal cord is involved, partial or complete loss of sensation and movement below the level of the injury occurs. The patient is usually severely shocked and exhibits all the signs of this condition.

With the possible exception of arterial haemorrhage fracture dislocations of the spine require more immediate care and management than any other injury, in other words the so called 'first aid' treatment is of vital importance. If the spinal cord or intraspinal nerves have already been damaged further injury to these structures must be avoided. If they have escaped, they must not be injured by incautious movement of the patient. The spine is unstable at the site of the injury, and this area must be protected from movement and stress. The patients must be transported on a rigid stretcher on to which they are placed with the utmost care, preferably laid face down to avoid any danger of spinal flexion.

When the patient has reached hospital, the diagnosis is confirmed by radiology and the extent of the damage is ascertained. The bodies of one or more vertebrae are usually wedged and the articular processes are either fractured or actually dislocated, the lower process of the vertebra above having over-ridden the superior process of the vertebra below.

The subsequent management of these fracture-dislocations depends upon whether or not there is associated damage to the cord or intraspinal nerves.

If there is no such damage the dislocation may be reduced either by manipulation or by open reduction or in certain circumstances the spine may be fused in the dislocated position and thus rendered stable.

If the cord or nerves are damaged, the difficulties of treatment are greatly increased. Although such damage usually occurs at the moment of injury

and may well be irreversible, pressure or traction on these structures should be relieved by accurate reduction of the dislocation in order to facilitate any recovery which may be possible. Because of the loss of sensation, prolonged pressure on any skin area is to be avoided and this precludes the use of plaster and makes the problem of nursing extremely difficult. By far the best method of treatment is immediate open reduction of the fracture-dislocation, followed by internal fixation of the spine by metal plates clamped to the spinous processes. This procedure renders external fixation unnecessary and greatly simplifies subsequent nursing. The best conditions for subsequent recovery of cord and nerve function are thereby established and the patient is made far more comfortable. Immobilization is continued until the spinal fractures have united and the soft tissues healed and, thereafter, if there is no nerve or cord involvement, muscle exercises and movements are practised to restore function. The management of patients with permanent spinal cord injury is extremely complex and is best carried out in one of the special spinal centres established for this purpose.

Subluxation of the cervical spine

The cervical region is more vulnerable than other parts of the spine, and injuries to the neck involving the spinal column are not uncommon in sport. The most common mechanism in these injuries is forced flexion such as might occur in a fall or a dive into shallow water. Lateral flexions and rotatory twists also occur in certain circumstances.

Subluxation of the cervical spine is the commonest flexion injury. A mechanical force either falls on the flexed spine or flexes it beyond its normal range of movement. The posterior ligaments, the supraspinous and interspinous ligaments, the capsules of the posterior articulations and the posterior common ligament are ruptured at some point and at this level displacement of one of the vertebral bodies takes place, but does not progress to actual dislocation of the posterior articulations or fracture of the vertebral bodies. This displacement only occurs when the spine is flexed, on extension the vertebral bodies resume their normal relationship. Subluxation of the cervical spine is rarely associated with injury to the spinal cord but some degree of traction of the cervical nerves at the level of injury is not uncommon. This stretching produces pain or abnormal sensations in the form of tinglings or 'pins and needles' in the area supplied by the sensory part of these nerves, and some weakness in the muscle groups enervated by their motor fibres.

The recognition of this fairly common injury is very important. Unless it is diagnosed and adequately treated, healing does not take place and the spine

remains unstable in flexion with associated symptoms and disability. The patient with a subluxation of the cervical spine gives a history of flexion injury followed by pain in the neck, this pain being aggravated by movements of the head and sharply aggravated by forward bending of the neck. There may be tingling or pain referred to one or both arms. On examination there is spasm of the cervical muscles and limitation of with pain on movements of the neck. When nerve root symptoms are severe there may be some sensory changes or muscular weakness in the hands or arms.

Radiographs taken in the ordinary manner show no abnormality as the vertebrae resume their normal relationship when the neck is in the neutral position and it is this apparent normality on X-ray examination that is the main reason why this injury so often remains undetected. The subluxation can only be demonstrated by lateral views taken with the neck in full flexion. Such radiographs clearly show any 'step' or break in continuity in the line of the vertebral bodies, and no radiological examination of the neck after injury is adequate unless such views are taken.

When a subluxation of the cervical spine has occurred the neck must be immobilized in the neutral position until the torn ligaments have healed. Immobilization can be effected by plaster or by the use of one of the various types of cervical collars of which the most comfortable and efficient are those of light polythene made to measure to fit the individual patient. The neck should be immobilized for about three months and during this time static contraction of the cervical muscles may be carried out to prevent loss of muscle bulk and power. When healing is judged to be complete on clinical and radiological examination the collar is discarded gradually, being left off for longer and longer periods. At the same time gentle extension, lateral and rotation exercises of the neck are carried out but flexion exercises should be avoided. If treatment is adequate these subluxations are not the source of any permanent disability.

Cervical dislocations and fracture-dislocations

The same forces which produce subluxations of the cervical spine can also, when they are sufficiently great, produce dislocations or fracture-dislocations. Such forces are not necessarily purely in the flexion plane but may be partly lateral or rotatory so that the dislocations or fracture-dislocations produced are correspondingly asymmetrical. Unlike similar injuries in the lumbo-dorsal region, in the neck a true dislocation can occur without any associated fracture of the vertebral bodies or articular processes. Various rather complex varieties of both fractures and fracture-dislocations occur at the atlanto-occipital and atlanto-axial joints. Both dislocations and fracture dislocations

are commonly associated with damage to the spinal cord and spinal nerves in their intraspinal course. The effects of complete interruption of the conductivity of the cord depends on the level at which it occurs. In the lower regions of the neck, complete paralysis and loss of sensation affects the entire trunk and all four limbs, a quadraplegia. Above the level of the third cervical segment the nerve supply to the diaphragm is cut off, respiration ceases and death occurs quickly. If the upper part of the cord is disrupted, as in judicial hanging, death is instantaneous. Cord damage may, of course, be partial with a corresponding variation in its effects. The effects of root damage also depend on the level and extent of root involvement, sensory and motor changes being produced in one or both arms.

The patient with a dislocation or fracture-dislocation of the neck gives a history of injury followed by acute pain in the neck, inability to move the head and, if the cord or nerves are involved, loss of power or weakness of some part of the body. If the cord is injured at a high level death may occur rapidly, or even instantly. There is usually obvious deformity of the neck with local pain and loss of movement. Neurological changes, if present, are easily detected.

The diagnosis and exact nature of the injury are confirmed by radiographs.

The treatment of cervical dislocation or fracture-dislocations is, like that of similar injuries elsewhere in the spine, made much more difficult if cord damage is present. The initial handling of the patient is, of course, important and any movement of the neck must be avoided. Reduction of the fracture or fracture-dislocation is usually effected by controlled skeletal traction applied through the skull. By varying the force and direction of the traction under radiological control, most dislocations or fracture-dislocations can be reduced in two or three days. Occasionally traction must be combined with open operation to free the interlocked articular facets. When reduction is complete the neck is immobilized in a suitable collar until healing takes place. Provided reduction is accurate and no permanent damage to the cord or nerves has occurred, the ultimate prognosis in these injuries is good and they are not usually associated with any permanent disability. The management of patients with cervical cord injuries is highly specialized, being even more difficult than when the cord is injured at a lower level. Such patients are best treated in a special centre.

5

INTERVERTEBRAL DISC LESIONS

CERTAIN pathological changes or, more accurately, a specific cycle of changes, which occur in the lumbar or cervical intervertebral discs constitute a clinical entity. The only term which may correctly be applied to this condition is a lumbar or cervical disc lesion. This name may be unsatisfactory in some respects but it is much better than the others in common medical and lay use, most of which are either meaningless or vaguely descriptive of one single episode in the cycle. Strictly speaking disc lesions are almost certainly not due to trauma and certainly they are not an immediate result of a single accident. Certain phases in the cycle are, however, precipitated or influenced by mechanical forces and for this reason disc lesions are often influenced by injury. Lesions of the lumbar intervertebral discs are common and are probably one of the most frequent of all sources of disability in athletes. For this reason a brief consideration of these lesions is necessary.

THE STRUCTURE OF THE INTERVERTEBRAL DISCS

The adult intervertebral disc consists of three basic parts. Above and below cartilage end-plates cover the central part of the vertebral bodies and form the upper and lower boundaries of the disc. The fibres of the annulus and nucleus arise from and are blended with these plates. The nucleus pulposus is the central part of the disc. This nucleus is of semigelatinous consistency and is composed of a loose net-work of fibrous tissue strands in the meshes of which are spindle-shaped fibrous tissue and cartilage cells. The whole nucleus is highly plastic, obeys the law of fluids, that is to say it is incompressible, and is held in shape by the surrounding annulus and cartilage end-plates. The nucleus constitutes rather less than one-quarter of the total volume of the disc. The annulus fibrosus is the outer layer of the disc. In the lumbar region it consists of ten to twelve fibro-elastic lamellae whose fibres run obliquely between their attachments to the cartilage plates and epiphyseal rings of the vertebrae above and below. The fibres of the different layers lie at an angle to each other and the annulus is weakest and most loosely attached posteriorly. The annulus acts as a retaining envelope for the nucleus by which its fibres are normally slightly distended. There is, however, no clear line of

demarcation between the annulus and nucleus, these structures merge one into the other and with increasing age the difference between them becomes progressively less marked.

THE FUNCTION OF THE LUMBAR INTERVERTEBRAL DISCS

The discs are an essential part of the vertebral column; they contribute to the length and shape of the spine, they participate in the transmission of weight and they supply the vital element of flexibility.

Movements of the spine as a whole are made up of a series of movements between the individual vertebrae, these intervertebral movements being greatest where the discs are thickest, that is in the cervical and lumbar regions. The nucleus acts as a fulcrum on which movement between contiguous vertebrae takes place and the annulus because of its elasticity and the direction of its fibres permits such movement and becomes taut at its extremes, acting as a check ligament (*Figure* 1).

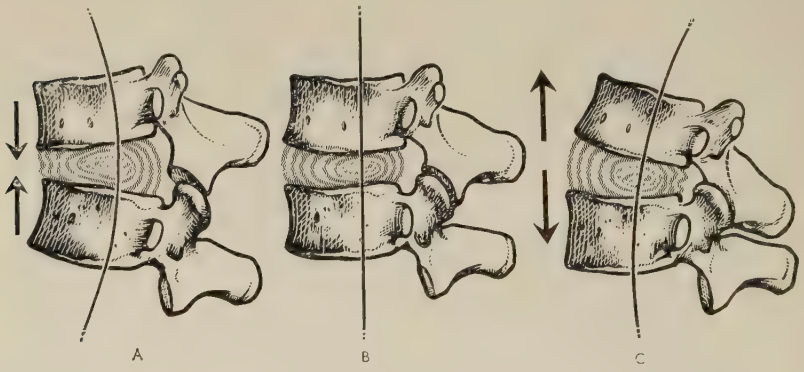

FIG. 1. The nucleus pulposus acts as a fulcrum on which movement between contiguous vertebrae takes place. The elastic fibres of the annulus, in which the nucleus is contained, becomes taut at the extremes of the various movements.

The intervertebral discs also have an important shock absorbing mechanism. When subjected to a compressing force the incompressible nucleus flattens and further distends the elastic annular fibres which then absorb part of this force. (*Figure* 2).

When the spine is compressed it is essential that the stresses be spread evenly over the surfaces of the vertebral bodies to prevent local pressure changes. The nuclei obey the law of fluids and transmit force equally in all

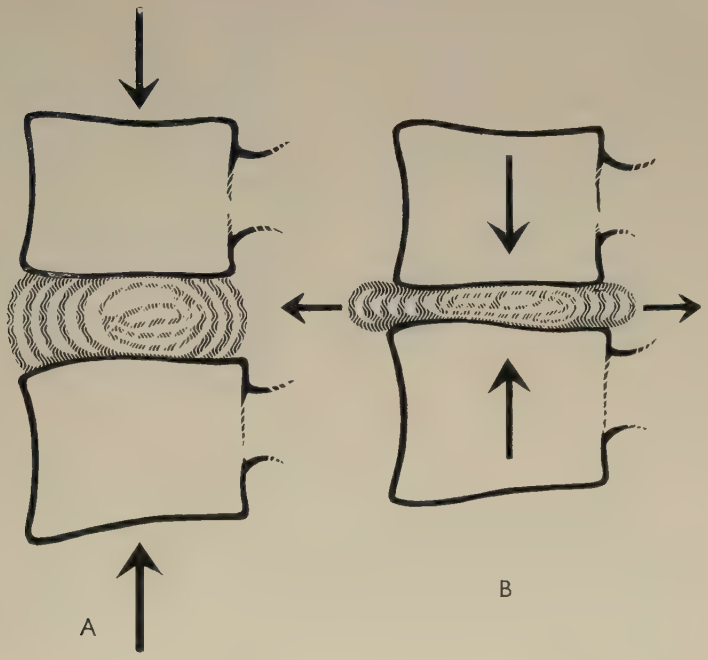

FIG. 2. The intervertebral discs act as shock absorbers. When the vertebrae are subjected to a compression force (A), the incompressible nucleus alters its shape and flattens, further distending the elastic fibres of its containing annulus. These fibres thus absorb part of the compressing force (B).

directions, ensuring that it is evenly distributed over the vertebral end-plates. The cartilage end-plates themselves protect the vertebral bodies from pressure changes. (*Figure* 3).

THE PATHOLOGICAL CYCLE IN LUMBAR DISC LESIONS

The changes which take place in the lumbar intervertebral discs occur in three stages. Each stage is associated with characteristic signs and symptoms which may be primary, that is due to the abnormal state of the discs themselves, or secondary, when they originate from other structures, notably those occupying the spinal canal, which become involved in the disc changes.

First stage

This consists of a progressive softening and breaking up first of the nucleus

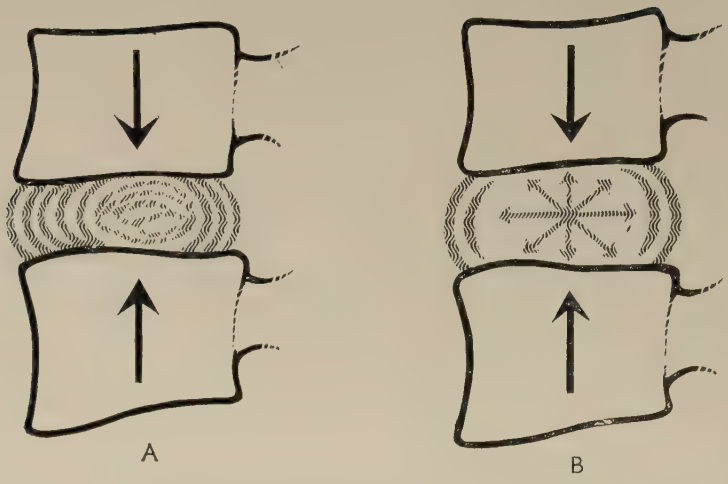

FIG. 3. The intervertebral discs ensure the even spread of compression forces. When subjected to such a force (A), the nucleus obeys the law of fluids and transmits it equally in all directions (B), so that it is spread evenly over the annulus and the cartilagenous end plates of the vertebral bodies.

pulposus and later of the posterior annulus of the affected disc. The nuclear changes progress to complete fragmentation when the nucleus is reduced to a varying number of separate detached pieces lying loose in semi-fluid material. At the same time the fibres of the posterior part of the annulus soften and disintegrate until finally the annulus ruptures and gives way, usually over a fairly small area to one or other side of the midline. The weakened annulus may rupture suddenly as the result of some strain or disintegrate gradually. The first stage ends with annular rupture and this stage may be described as a progressive softening and final bursting of the disc. (*Figure* 4).

Second stage

When the annulus has ruptured, broken-up nuclear fragments are squeezed out posteriorly through the annular defect. A normal nucleus is anchored in position by its fibrous tissue framework and does not displace to any great extent when a hole is cut in the annulus but degenerate nuclear tissue, being broken up and fragmented, is quite readily extruded by the positive pressure to which the intervertebral disc is subjected at all times. When the annulus has given way the posterior longitudinal ligament is the only structure which

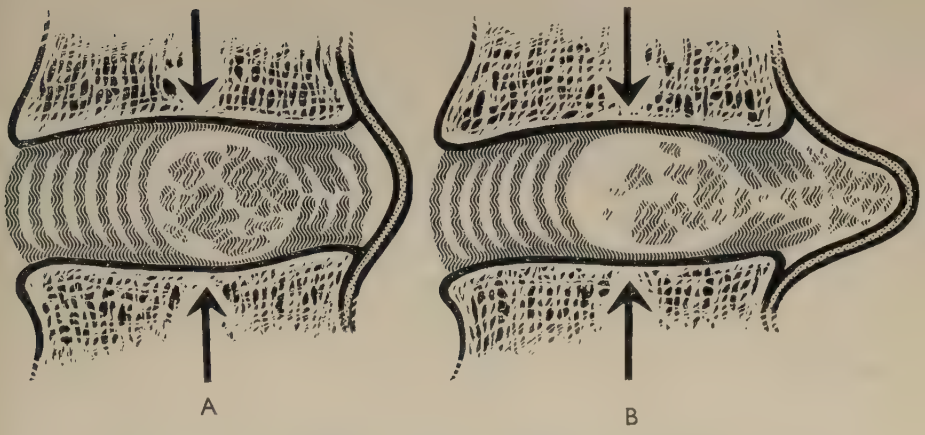

FIG. 4. The pathological cycle which constitutes a lumber disc lesion takes place in three stages. The first stage consists of a progressive softening and distintegration of the nucleus and posterior part of the annulus (A) and ends in annular rupture. The second stage is that of nuclear extrusion. Fragments of the broken-up nucleus are forced backward through the annular defect and form a tumour which projects into the spinal canal (B). This tumour is usually contained by the posterior longitudinal ligament but sometimes this ligament itself may disintegrate, allowing seqestra to escape freely into the canal.

prevents the free escape of displaced nuclear material into the spinal canal. This ligament is not strong enough, however, to retain nuclear substance between the vertebral bodies. It becomes stretched and lifted from its attachment so that the displaced nuclear tissue forms a protrusion or tumour projecting into the spinal canal itself. Occasionally the ligament ruptures over the summit of this tumour and then free nuclear fragments escape into the canal itself. The size, shape and site of nuclear protrusions differ and these variations, in association with the normal differences in the size and shape of the spinal canal, are responsible for the difference in the nature of the secondary symptoms produced at this stage.

Nuclear extrusion is an intermittent phenomenon and the process continues off and on over a period of months or years. Various factors are responsible for its intermittent nature. Nuclear disintegration is usually not complete when annular rupture takes place, the fragmented portions are extruded and this process then ceases until more nuclear tissue breaks up, when further extrusion may occur. The annular defect is sometimes plugged by a large nuclear fragment but this is a temporary phenomenon. Such fragments ultimately escape or, in favourable circumstances, slip back between the verte-

bral bodies; and these events cause variation in symptoms from time to time. The forces causing extrusion reach temporary equilibrium. Normally the nucleus is under positive pressure of varying degree at all times and slightly distends the elastic annulus by which it is contained. With annular rupture and the escape of nuclear tissue, the vertebral bodies come together until they rest on the annular tissue which prevents further approximation and resists the compression force. At this stage equilibrium is reached and persists until the compression force is increased by muscular effort, exertion or movement. When this happens more nuclear tissue may be extruded.

The second stage only ends either when healing of the damaged disc has taken place or when the entire nucleus has disintegrated and been extruded.

Third stage

This stage is a slow process of healing and consists of fibrosis and repair of all the damaged disc tissue. The third stage overlaps the second, once the annulus ruptures fibrosis and healing begin in both the annulus and in the abnormal nuclear tissue. The healing changes progress slowly, being interrupted from time to time by fresh episodes of nuclear extrusion or by further damage due to mechanical stresses. Gradually, however, the disc tissue dehydrates and shrinks and the intervertebral space slowly becomes narrower. There is no longer any normal disc function and pressure changes often take place in the bony vertebrae, a firm stable fibrous ankylosis is ultimately established and the damaged intervertebral joint is then mechanically sound. This may be regarded as the end stage of healing but, of course, the ankylosis

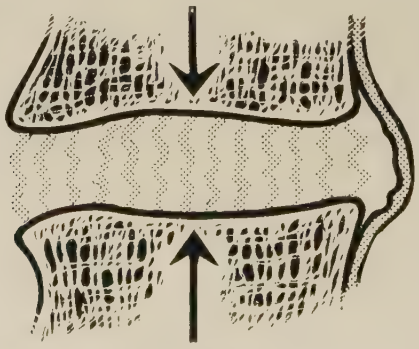

FIG. 5. The third stage is that of fibrosis and repair. The damaged nucleus and its annulus is slowly converted into fibrous tissue which ultimately replaces the entire intervertebral disc and binds the vertebral bodies together.

is always fibrous as opposed to bony and is susceptible to damage by excessive mechanical stresses. (*Figure* 5).

SITE OF LUMBAR DISC LESIONS

Most lesions occur in the lower two lumbar discs, the upper three being very much less frequently affected. Two discs, usually the lowest two, may be affected simultaneously. The Author's figures in one thousand consecutive cases proved at operations were:—L5/S1 lesions 46·8 per cent, L4/L5 lesions 40·4 per cent, L1/L2, L2/L3 and L3/L4 lesions 2·1 per cent and double lesions 10·7 per cent.

AETIOLOGY OF LUMBAR DISC LESIONS

We do not know with absolute certainty why lesions of the type described affect the intervertebral discs. Certain facts are, however, indisputable.

True lesions are almost entirely confined to the lower lumbar and lower cervical discs and these regions of the spine are unique in two respects. Firstly, they are the site of the secondary spinal curves. These curves develop to meet the demands of the erect posture and are produced by a relative increase in the anterior depth of the intervertebral discs themselves in contrast to the primary curves, which are due to the shape of the vertebral bodies.

Secondly the discs are thickest in these regions and this increase in disc depth is associated with a local increase in the overall mobility of the spine.

It is probable that progressive degeneration of the lower lumbar and cervical discs occurs because of the special mechanical demands made on them as the result of man's adoption of the erect posture. It may seem a long time since we came down from the trees to walk upright on the ground, but the process of adaption is still not complete.

While disc lesions may be a degenerative process occuring as the result of wear and tear other factors must be considered.

Of these injury is the most important. Formerly all disc lesions were considered to be purely traumatic, that is the immediate result of an injury of some kind. This is obviously incorrect. About 40 per cent of patients with typical lesions give no history of accidental injury while in others the strains associated with the onset of symptoms are quite trivial in nature. It is true that in many people there is a dramatic connection between a strain of some kind and the immediate onset of severe pain and such people are often con-

scious of something 'snapping' or 'giving way' in the back. It is highly unlikely, however, that in such cases the affected disc was normal before the incident to which symptoms are attributed. Once the cycle of pathological changes has started the affected disc is weakened and may be damaged by strains which would not affect a normal disc. Quite often the effects of such a strain may be dramatic in nature, causing sudden changes both in the disc itself and in the symptoms produced. For example, final rupture of the degenerate annulus at the end of the first stage is often produced by flexion strain, during the second stage episodic nuclear extrusion depends on the compression forces to which the disc is subjected while during the third stage the fibrous tissue formed during the healing process may be stretched or torn if subjected to excessive strain. Injury, therefore, probably only produces immediate effects in discs which are already abnormal.

It is impossible to be equally confident about the late effects of trauma. It may be that a single severe injury can be followed by the gradual onset of degenerative nuclear changes which may progress to produce the cycle associated with a typical disc lesion.

Other factors have been considered as possible causes of disc lesions. Compression stresses, imbibation of fluid, mental stress, focal sepsis, pregnancy, nutrition and, perhaps the most important, the inherent quality of the fibrous and elastic tissue in the individual concerned may each play some part.

SYMPTOMS OF LUMBAR DISC LESIONS

Clinically the picture associated with lumbar disc lesions is that of intermittent attacks of low back pain often accompanied by pain referred to one leg. These attacks are often precipitated by flexion strain and are aggravated by exertion and relieved by rest. The condition is markedly episodic, the attacks being punctuated by periods of remission which may last for weeks or months. The disorder is chronic, the patients troubles recurring over a period of years. Not all disc lesions are associated with a classical clinical picture, however, there are many variations which may occur as the result of changes in the pathological cycle which produces these lesions.

Essentially there are two groups of symptoms, primary, which arise from the abnormal disc itself and from the intervertebral joint of which the affected disc is an integral part and secondary, which arise from other structures, notably those occupying the spinal canal, which become involved in the disc changes.

The primary symptoms are referred to the low back itself and consist of

pain, and in some instances, alterations in posture. About 90 per cent of patients complain of low back pain at some time or other and the nature of the pain varies with different stages of the cycle. During the first stage of nuclear disc degeneration it takes the form of aching in the low back which is aggravated by exertion and relieved by rest. Occasionally this aching becomes moderately acute and incapacitating and this is probably associated with imbibation of fluid, that is swelling of the abnormal nucleus. Rupture of the annulus at the end of the first stage is associated, often dramatically, with an acute exacerbation of symptoms due to extrusion of nuclear sequestra. During the whole of the second stage these exacerbations continue whenever nuclear extrusion takes place. From time to time nuclear fragments become 'locked' between the vertebral bodies and produce severe and completely incapacitating pain. This persists until the fragment is either extruded completely or slips back within the concavity between the intervertebral bodies. The onset of this 'joint locking' is often dramatic and, in some instances, the relief may be almost as immediately dramatic. Between the episodes of nuclear protrusion the second stage is associated with back ache of varying degree, again made worse by strains and relieved by rest. During the third stage the low back symptoms depend on the nature of the healing tissue and the strains to which it is subjected. In the early stages, when the tissue is soft and easily stretched, low back pain occurs as the result of any strains and only subsides slowly with rest. Later, as the fibrous ankylosis becomes more stable, the joint is less affected by exertion and symptoms produced by strains are less severe. Ultimately, when the ankylosis is stable, the joint becomes pain-free. A small percentage, perhaps about ten per cent, of patients with lumbar disc lesions never complain of any pain in the lumbar region. In these people the clinical findings in the low back are typical and secondary symptoms develop in the ordinary way, but presumably because of defective innervation of the lumbar discs and low lumbar joints, they have no pain arising from these structures. Such patients may be difficult to treat because of the absence of premonitary symptoms.

When the intervertebral joint becomes 'locked' by a nuclear sequestrum or when a root in its extrathecal intraspinal course becomes displaced by a nuclear protrusion alterations in posture which cannot be voluntarily corrected by the patient may occur. These take the form either of a fixed lumbar flexion, a lumbar scoliosis or a combination of these two.

The secondary symptoms produced by lumbar disc lesions are mainly those of involvement of structures occupying the spinal canal. Of these structures the spinal roots in their extrathecal course are the most commonly affected. These roots, at the lower lumbar level, cross the intervertebral discs

from above downwards before they leave the spinal canal and are in close contact with the posterior longitudinal ligament covering the posterior surfaces of the discs themselves. Nuclear extrusion commonly takes place in the areas in contact with the roots, and the nuclear tumours so produced stretch the roots themselves. This stretching produces pain referred to the distribution of the affected root and is the basic cause of the sciatic pain associated with disc lesions. There is also some disturbance in root function with a little muscle weakness and some diminution of sensation in the skin areas supplied by the root involved. Occasionally when a nuclear extrusion is large and the spinal canal is relatively small and, as often happens, trefoil in shape with a 'lateral recess', the root is actually compressed between the nuclear tumour and the bony wall of the canal. The compression produces an immediate loss of root function often without any associated pain. This phenomenon occurs in the occasional patients who have severe muscle weakness and areas of complete anaesthesia in the dermatomes corresponding to the affected root without associated pain.

Very occasionally a massive sequestrum occupies the whole of the spinal canal and compresses all the roots of the cauda equina in their intrathecal course. Cauda equina compression is associated with severe and widespread weakness and loss of sensation in the legs and disturbance of function of the bladder and rectum. Unless such pressure is relieved quickly these changes may be permanent.

In the third stage the extrathecal roots may become adherent to the posterior surface of the disc at the site of annular rupture. Normally these roots are mobile and move fairly freely in association with movements in the leg. If the roots are bound down such movement may cause stretching and sciatic symptoms.

Not all patients with disc lesions and nuclear extrusion have sciatica. If the site of the annular rupture is central the nuclear tumour may never come into contact with the extrathecal roots, and if the canal is large and the roots comparatively lax tumours which do come into contact with these roots may not stretch them sufficiently to produce symptoms.

THE SIGNS (CLINICAL PICTURE) OF LUMBAR DISC LESIONS

The diagnosis of a lumbar disc lesion is not difficult provided the examination of the patient is sufficiently thorough. It is true that many other conditions produce low back pain and a number of these are associated with pain referred to the legs but neither the clinical picture nor the history associated with these conditions simulates disc lesions exactly.

Examination of the lumbar spine

The findings on examination of the patient with a lumbar disc lesion are usually quite typical in the acute phases, particularly when nuclear extrusion has recently occurred. The posture of the patient is often unmistakable; the lumbar spine is held rigid in a flattened position with the dorsal and cervical spines slightly flexed, the whole body is bent forwards at the hips and the affected leg is held with the hip and knee in flexion and the ankle in plantar-flexion, only the toes resting on the ground. A spinal scoliosis is often present and this is obvious, one hip and one shoulder being unduly prominent. The patient finds it difficult to stand up or sit down and the whole picture is that of a person in acute discomfort due to severe low back pain. On examination of the lumbar spine during the less acute phases, the findings are muscle spasm in the lumbar region, flattening of the normal lumbar lordosis, a varying amount of postural scoliosis which may be made more obvious by flexion, and limitation of movement, flexion and extension being more affected than lateral flexion and rotation. There is often local tenderness at the site of the disc lesion and percussion of the slightly flexed spine produces pain both in the back and referred to one leg.

Examination of the legs

On examination of the legs it is found that sciatica is usually mono-radicular in distribution (occasionally, in special circumstances, two roots may be involved either because a double lesion is present or because of migration of a nuclear tumour under the posterior longitudinal ligament). There may be diminution of sensation over part or all of the dermatome supplied by the affected root, and some weakness and even wasting of the muscles innervated by this root. Straight leg raising is diminished on the side of the affected root and to some extent on the opposite side and the allied tests are also positive.

Reflex changes are present if the fourth lumbar or first sacral roots are involved. Coughing or sneezing or anything else which raises the intrathecal pressure causes an increase in the patient's pain.

The only ancillary investigation necessary in the examination of all patients with a lumbar disc lesion is X-ray of the spine. In the later stages of such lesions there may be intervertebral narrowing at the site of the affected disc, but until this stage is reached the radiological appearance of the lumbar spine is normal.

TREATMENT OF LUMBAR DISC LESIONS

Probably more arrant nonsense is talked and practised in the treatment of lumbar disc lesions than in the management of any other condition. The simple truth is that once nuclear degeneration has occurred the replacement of the damaged nucleus by fibrous tissue is the only possible form of natural healing. If the nuclear damage is widespread or if rupture of the annulus has occurred such healing necessarily involves a fibrous ankylosis of the affected intervertebral joint and it is only by the formation and stability of such an ankylosis that ultimate recovery can be achieved. The aim of treatment is, therefore, to establish the best possible conditions for such healing, that is to protect the damaged joint from mechanical strains and from movement. The only way in which this can be done is by curtailment of the patient's activity and immobilization of the lumbar spine.

These basic measures, rest and immobilization, are the sheet anchors of treatment. Rest may take any form from complete immobility in bed in the supine position to a simple avoidance of flexion of and flexion strains on the lumbar spine. Immobilization may be achieved by the use of a plaster bed, plaster spica, plaster jacket or some form of lumbar corset. These two measures should be employed in the management of all patients with lumbar disc lesions, their exact forms being determined by the nature and stage of the lesion itself.

In certain circumstances, notably when nuclear sequestra are causing pressure or embarrassing healing, conservative measures are not enough. When this happens the sequestra and the remainder of the nucleus must be removed surgically and such an operation relieves pressure symptoms dramatically and establishes the best possible conditions for subsequent ankylosis of the damaged joint. In order to ensure that this ankylosis takes place, however, the basic principles of treatment must be applied after operation and the joint protected for a sufficient period of time to ensure stable healing. Occasionally it is desirable to speed up or to make certain of stable ankylosis by surgical means, that is by arthrodesis of the damaged joint.

The basic principles might seem simple enough although their application to individual patients is a matter requiring very considerable judgement and experience. Nevertheless, probably because of the apparently irradicable belief that when a disc is abnormal something has 'slipped' or 'displaced' and must be 'put back', many measures are employed in the management of these conditions which not only can do no permanent good but do in fact cause further damage to the affected disc and intervertebral joint and delay or even prevent natural healing

DISC LESIONS IN ATHLETES

Once a disc lesion has occurred and the pathological cycle has become established there is no quick way in which a 'cure' may be achieved. Obviously the application of the principles of rest and immobilization are going to involve curtailment of activity to a greater or lesser degree for some time. For this reason the occurrence of a true lesion in a professional or first-class amateur athlete is a very grave matter and almost invariably is going to be associated with a considerable period of disability and some loss of efficiency. Yet there is no short cut which may be employed in their management and the basic principles of treatment must be applied if severe and permanent disability is to be avoided.

Certain things can, however, be done to prevent the occurrence of disorders of the lower lumbar discs, even in those who engage in strenuous athletic pursuits. If three simple principles were observed such lesions would occur very much less commonly than they do. First, the athlete should be trained to adopt a good posture at all times, the active alerted posture, once it becomes habitual, prevents mechanical stresses and damage to soft tissues all over the body, and in particular spares the lower lumbar discs from much unnecessary strain. The importance of this posture in the promotion of physical well-being, not only in athletes but in everyone else, cannot be over-emphasized. Secondly, the athlete should be provided with his own built-in lumbar support in the form of strong muscles. The abdominal muscles and the extensor muscles of the spine should be developed by appropriate exercises until they can protect the spinal joints and soft tissues against any mechanical strain likely to be encountered; and the building up of these muscles is a vital part of the training of any athlete. Finally, in each individual sport, scientific coaching is vitally important. No physical action which throws a sudden severe strain on the lower lumbar spine can be efficient, and proper coaching will prevent the athlete from damaging his low back unnecessarily and to no purpose. If a disc lesion has occurred in an athlete these three principles, in conjunction with the treatment already outlined, will promote recovery and prevent subsequent damage to the abnormal disc. A good posture, strong muscles and avoidance of unnecessary strains on the low back may enable an athlete who has suffered from a disc lesion to continue active sport for years without trouble.

INDEX

Abdomen, strains, after inactive slumping position, 70
Abrasions, in baseball, 153
in basketball, 156, 158
in boxing, treatment, 160
calf, in rowing, 228
Absorption, bone, at fracture surfaces, 489–90
Acclimatisation, arrival early for meetings, 88
cold, 49–52
heat, 47–9
air, 49
water, 49–52
warm-up, 52–3
Achilles tendon, *see* Tendon, Achilles
Acroparaesthesia, and slumping posture, 70
Action potential, 20
Adhesions formation, *see* specific injuries
broken by manipulation under general anaesthesia, 272, 305
reaction, 305
Age, and bellows function of lungs, 38
deterioration according to, 290–1
effects, 290–1
on maximum performance, physiological aspects, 57–9
and healing of fractures, 484–5
Ageing, fibrocystic changes, 281
influence on athletic injuries, 281–2
Alcohol, before an event, 129
during training, 127
Alveolar capillary function, 38
American Water Ski Association, 242
Anaesthesia, examination under, 273
manipulation under, 304–5
Analgesics, with injection therapy, 315
for pain, 316
Ankle, *see* Joints, ankle,
Antecubital swelling, in baseball, 150
Anterior compartment syndrome, in baseball, 152
Anterior tibial syndrome, 354
Anticoagulants, effect on fracture healing, 485
Arm, fractures, 524–30
upper, biceps injuries, treatment, 382, 391–2
see also Forearm; Joints, elbow; Limb(s)
Arterial spasm, and fractures, 487
Arteries, disorders, 356–7
and activity, 341
injuries, treatment, 339–58
ligation, 348
thrombosis, 354
see also Thrombosis
trauma, first aid treatment, 341–2
see also Blood supply; Blood vessels; Volkmann's Ischaemic Contracture
Arteritis, crutch axillary, 352
Arteriograms, 272
Arthritis, and inactive slumping posture, 69
Arthrodesis, for osteoarthritis, 466
Arthrograms, 271
Arthrography, air, 429–30
Arthroplasty, for osteoarthritis, 466
Articulations, intervertebral, structure, 556
Astragalus, fractures, 513
Athlete, outstanding, 108
qualities, 106
Athlete's foot, in swimmers, 241
Athletes, injuries to, 142–7
and physiologist, different approaches to training, 88
see also Runners, Sprinters, etc.
Athletics, physiological and psychological approach, 105–12
and somatotyping, 94–104
Atlas and axis, articulation, 566–7
Atrophy, Sudeck's, 492
Attachments, muscle, 11–13, 14
Axis and atlas, articulation, 566–7
Axonotmesis, 336–7
prognosis, 339

Back, 'broken', 583
low, disorders, and golf, 134
pain, and lumbar disc lesions, 600–2
strained, in fencing, 177
Backache, and draughts, 285
Balance, 24–7
organs of, 24
Ball, cricket, damage from, 168
hockey, 206–7
Bandaging, hand, in boxing, improvements, 166
Bannister, R., 103
and training for peak performance, 85
Baseball, elbow injuries, 149–50, 153–4
'finger', 151
fractures, 152–3
head injuries, 153
injuries in, 148–54
joint injuries, 152–3
maxillo-facial injuries, 153
motor power unit injuries, 151–2
overload injuries, 151–2
scapula injuries, 148
shoulder injuries, 148–9
soft-tissue injuries, 153
Basketball, injuries, 155–8
ankle, 155–6
concussion, 157
finger, 156
fractures, 156
knee, 156
muscle-tendon-bone unit, 155
prevention, 157–8
muscle, 156
soft-tissue, 156
Baths, after injury, 449
Turkish, 85
Beaters, and prevention of accidents during shoot, 198
Bending, mechanics, 73–5
muscles involved, 73–5
Bennett fracture of thumb in boxing, 164
Biceps, *see* Muscles, biceps
Bindings, release, on skis, 256–7, 260
Black-eyes, *see* Haematomas
Blade, fencing, force and stresses, 179, 183–8
Bleeding, venous, control, 349, 350–1
Blisters, in rowing, 227
treatment, 227
in running, 147
Blood loss, replacement, 342
Blood supply, and healing of fractures, 476–9
see also Arteries, Veins,
Blood vessels, arterial spasm, cause and treatment, 348
associated with fractures, 487–8
contusion, treatment, 348
disorders, complications of injury, 462
examination for injury, 279
ligation, 348
in relation to injury, 264–5
repair, 342–6
thrombosis, after strenuous activity, 353–5
and time lag of repair, 352
traumatic division, 346–52
complete, 347–8
grafts, 347–8
partial, 346–7
see also Arteries, Veins
Blood volume, increase during training, 45
Body build, *see* Somatotype
Body structure, *see* Structure
Bodies, loose, in shoulder, baseball, 149
Bone(s), architecture, 4
composition, 4
injury, examination, 270
in football, 139
necrosis, avascular, in fractures, 490–2
purpose, 3–4
see also Fractures, Muscle-tendon-bone unit and names of individual bones
Boutonniere deformity, in baseball, 151
Bowler (cricket), correction of foot pain, 78
and muscle tears, 134
Boxing, abrasions in, 160–1
concussion, 161–4

Boxing—cont.
deflected septum in, 159–60
electroencephalograms in, 161–4
epistaxis, 159
eye injuries in, 161, 164
fitness in, importance, 165, 167
fractures of skull and jaw in, 164
haematomas in, 161
injuries, 159–67
statistics, 159
intracranial haemorrhages in, 164
lacerations in, 160–1
nasal bone fractures, 159–60
training, second week, 85
Brasher, Chris., and training for peak performance, 85
Breath-holding, 107
dangers, 56, 353
Breathing, maximum capacity, 37–8
oxygen, effects, 55–7
Bronchitis, and maximum breathing capacity, 38
Bruises, and buccal varidase, 165
in hockey, 207
see also Contusions, Haematomata
Buccal varidase, and reduction of bruises and haematoma, 165
Buerger's disease, *see* Thromboangiitis obliterans
Bursae, and diagnosis of injury, 264
injuries to, classification and treatment, 330–1
and knee joint injuries, 435
sub-acromial, calcification of walls, treatment, 331
Bursitis, acute, in baseball, 148
chronic, in baseball, 148
sub-acromial, treatment, 390–1
treatment, 331
Burst injuries, from shotguns, 195
Butazolidin and colchicine, treatment, calcified deposits in shoulder joint, 390

Caffein-containing drinks, and physical performance, 129
Callouses, on hands, of golfers, 205
Calorie(s), definition and interpretation, 125
requirements of athletes, 126
allowance during training, 83
Canal(s), semi-circular, 24, 25–26
spinal, structure, 557–9
Cap, cricket, to prevent injury, 168
Capsulitis of shoulder joint, deep x-ray therapy, 309
and slumping posture, 70
traumatic, manipulation under anaesthesia, 458–9
treatment, 395–7
Carbohydrate, calorie value, 125
food high in, 125
high content in meal before contest, 128
Carbon dioxide concentration, toleration, 52
Carbon dioxide tension in blood after oxygen, 56
Cardiac output, inadequate, and blood oxygen, 53
see also Heart
Cardiovascular function and oxygen transport, 40–6
Carpal bones, fractures, 526–7
Carpal scaphoid factures, 526
Carpal tunnel syndrome, and slumping posture, 70
Carpus, fractures in baseball, 152
Cartilage, articular, anatomy and physiology, 265–6
lesions, in golf, 202
lesions, in hockey, 208
retracting, diagnostic value, 424
semilunar, injuries, in basketball
Cerebellum, function, 24
Certificate, medical, before motor racing, 224
Chataway, C., and training for peak performance, 85
'Charlie horse', (muscle contusion) in baseball, 152, 156
Cheek, bruising, in shooting, 194
Chest, fractures, 531
injuries, in driving, 240
Chondromalacia patellae, 434
early diagnosis, 269
following knee injury, 425

Chymoral, 332
administration, oral, 316
Circulation, disturbance and diagnosis of injury, 268
effect of training on, 45
Claudication, intermittent, of calf, 356
Clavicle, fracture in hockey, 208
immobilisation, 531
injuries, 402–4
Clinics, treatment, 289–90
Clothing, and prevention of injury, 294
protective, worn by fencers, 178, 179, 181
suitable, 90
Coach, function, 109
and psychological approach, 110
and selection of potential athlete, 108–9
Coccyx, fractures, 522
injuries, 411
Colchicine, and butazolidin, treatment, calcified deposits in shoulder, 390
Cold acclimatisation, 49–52
air, 49
water, 49–52
effect on tissues, 285
see also Temperature, Weather
Collagen, of fibrous tissue, pathology, 282–5
diseases, following injuries, 467
Colles' fracture, 527
Collision injuries in football, 141
in hockey, 206, 208
in rugby football, 233
Column, spinal, and body movement, 569–70
cervical region, dislocations and fracture-dislocations, 591–2
treatment, 592
strains, in golf, 201–2
subluxation, 590–1
changes, and posture, 571
degenerative injuries in golf, 202
and development of posture, 574
discs, intervertebral, lesions, 593–605
dislocations, 587–92
effects on nerves and cord, 587–8
effect of gravity, 567–8
fracture-dislocations, 587–92
effect on nerves and cord, 587–8
fractures, 583–92
functions, 550
in water ski-ing, 247
joints, 'strains' and 'sprains', 579–84
lesions, traumatic, 575–592
ligaments, injuries, 578–9
mechanical forces acting on, 567–9, 571–2
movements, 561–70
between individual vertebrae, 563–6
correlation with other parts of the body, 569–70
dorsal region, 567
and posture, 561–74
of the spine as a whole, 566–7
muscle injuries, 575–8
and muscle tone, 567–8
and posture, 570–4
soft tissue injuries, 575–8
structure, 550–60
articulations, intervertebral, 556
canal, spinal, 557–9
discs, intervertebral, 552–3
length, 555
ligaments, spinal, 556
muscles, 559–60
overall shape, 553–5
vertebrae, bony, 551–2
distribution, 550–1
thoracic, strains, in golf, 201–2
see also Discs, intervertebral, Vertebrae
Concussion, in basketball, 157
in boxing, 161–4, 405
treatment, 161–2
in football, 139
in hockey, 208
in jockeys, 212
from knock-outs, 405
in motor racing accidents, 224–5
Congenital abnormalities, predisposition to injury, golf, 202
Constitution, and choice of athletics, 34

Contractions, muscular, anatomy, 13, 16
graduated, treatment by, 308
isotonic and isometric, 289–90
minimum number to maintain tone and volume, 308
Contracture, Volkmann's Ischaemic, *see* Volkmann's
Contusion(s), bone and cartilage, treatment, 331–2
difficult to make symptom free quickly, 453
in lower extremities, 414–16
common sites, 414
muscles, 416–8
muscle, posterior spinal, 410
treatment, 320–2, 416–8
pathology, 276
periosteal, anodal galvanism for, 308
of the scalp, 405
skin, treatment, 319–20
sprains of dorsum of hand, 364–5
thoracic wall, 406
of tissue of forearm, 370
treated by injection therapy, 314
vessel, treatment, 348
see also Haematomas
Coracoid, fracture, in shooting, 194
Cord, spinal, effects of dislocations and fracture-dislocations on, 587–8
Cortisone, effect on fracture healing, 485
see also Hydrocortisone
Cramp, muscle, treatment, 89
night, 355
Crepitus, and diagnosis of injury, 268
Cricket, injuries in, 168–72
return to after injury, according to position in field, 290, 293
see also Bowlers
Crutches, and axillary arteritis, 352
Curve, Intensity Duration, 271
in nerve injury, 334–5
Cuts, around eyes, in boxing, 166
treatment, 319
Cypress Gardens, Florida, and water ski-ing, 242
Cysts, dermoid and ovarian, removal, 116
Death, sudden in athletes, 353
see also Potassium
Deficiency diseases, and fracture healing, 485
Diagnosis of injury, examination, 268–70
history, 266–8
special investigations, 270–3
Diathermy, short-wave, faradism, surging, 308
galvanism, anadal, 308
interrupted galvanism, 307–8
Diet(s), crash, avoidance, 127
daily intake of foods, 128
during training, 126–8
immediately before contest, 129–9
overeating, 127
in relation to training period, 126
see also Nutrition,
Discs, intervertebral, cervical, lesions, in ski-ing, 252
degenerative changes, following inactive slumping posture, 70
lesions, 593–605
lumbar, function, 594–5
lesions, aetiology, 599–600
examination of legs, 603
examination of lumbar spine, 603
healing, 598–9
in rowing, 228
pathological cycle, 595–9
signs (clinical picture), 602–3
site, 599
in ski-ing, 252
symptoms, 600–2
treatment, 604
in spinal movement, 563
structure, 552–3, 593–4
Discograms, 272
Dislocations, cervical spine, 591–2
joint, treatment, 329–30
shoulder, associated injury, 393–4
causes, 393
complications, 395
reduction, and treatment, 394–5
see also other joints affected
spinal, 587–92

Diving injuries, 240–1
'Blast', 240
Drainage, fluid, measures to promote, 298
Draughts, and development of low back pain, 295

Ear, cauliflower, in boxing, 167
prevention, 405
injuries to, in rugby football, 231, 234
inner, and balance, 24–7
Ectomorphy, 94–104
'cerebrotonic' personality, 98
definition, 94
and games of skill, 101
E.E.G., *see* Electroencephalograms
Effusion, traumatic, cause, 275
causing pain, 316
in the fingers and hand, 359
pathology, 278–81
and tissue reaction, 282
see also Haematoma
Elastoplast strapping, uses, 300–1
Elbow, *see* Joints, elbow
tennis, *see* Tennis elbow
Electrodiagnosis, 271–2, 333–7
Electroencephalograms, in concussion in boxing, 161–2
performed on boxers after fights, 162–4
Electromyographs, 272
in nerve injuries, 335–6
Elliott, H., and training for peak performance, 85
Embolism, emboli, blood, associated with fractures, 488
fat, 456
fat, associated with fractures, 488
Emotion, *see* Psychological factors
Endomorphy, 94–104
definition, 94
'viscerotonic' personality, 98
Energy, and calorie requirement, 125
expenditure, and pulse frequency, 43
Enzymes, anti-inflammatory, oral administration, 316
Epee, points, 182–3
Epicondylitis, *see* Tennis elbow
Epistaxis, in boxing, 159
Equilibrium, *see* balance
Exercise(s) ability, maximal, 43
after fractures, 508
compared with faradic stimulation, 309
graduated, during training, 83–5
heavy, effect on blood oxygen, 54
oxygen, before and after and during, 56
relaxation, 86
and rest, in treatment, 288
specific, during training, 85–7
and venous thrombosis, 355
Exostosis, sub-glenoid, in baseball, 149
Eye, injuries in boxing, 164
cuts around, 166
damage by golf ball, 203

Face injuries, in rugby football, 231
in shooting, 194
Facilitation, proprioceptive neuromuscular, 310–12
Facio-maxillary fractures, 532
Falls, in riding, 211–2, 214–5
Faradism, after injury, critical survey, 451–2
surging, 308
'Fartlek Interval Training', 87
Fascial planes, and spread of haematoma, 263
Fat, body, and cold acclimatisation, 49–50
calorie value, 125
food high in, 125
pads, infrapatellar, contusions, 434
Fatigue, 'running through', 107
and ski-ing injuries, 257
and tennis elbow, 218 219
Feet, *see* Foot, feet
Felt and webbing support, 300
Femur, fractures, 521
associated with main vessel injury, 352
treatment, 521
stress, clinical findings, 545–6
site, 545
treatment, 546–7

Fencing, 173–190
fatal accidents, 179–81
analysis, 181–3
injuries, due to impact, 177
due to penetration, 178
due to reversal of force, 177
movements, 173–7
pulled muscles, 177
strained back in, 177
tennis elbow in, 177
terms, 190
Fibres, muscle, 11
arrangement, 13
nerve, structure, 16–20
Fibromyomata, occurrence in woman athletes, 119
Fibula, fractures, 517
stress, clinical findings, 541
prognosis, 541
site, 540
treatment, 541
head and neck, contusions, 415
see also Tibia and fibula
Finger(s) 'baseball', 151–2
circulation, 359
damage to during wicket keeping, 170, 171
dislocations, in rugby football, 234
fractures, 524
in catchers, 152
in diving, 240
injuries, in basketball, 156
in cricket, 169, 171, 172
little, injury in fencing, 178
mallet, treatment, 363
snapping (trigger), 363–4
splitting of skin, in bowlers, 170
trigger injuries, 193–4
trigger (snapping), 363–4
see also Joints, finger
First-aid, bag, contents, 342
treatment, principles, 294
vascular trauma, 341–2
Fitness, in boxing, 165, 167
and health, relationship between, 82
and training, 82–93
Flèche, in fencing, description, 175
Fluid, drainage, measures to promote, 298
liberal amounts during training, 127
oedema, and hydrolysis of collagen, 283–5
synovial, 265–6
aspiration, 295
see also Effusions, traumatic
Folds, alar, contusions, acute, 434
Food, *see* Diet, Nutrition, and individual headings
Foot, feet, care of, by athletes, 147
contused, x-ray treatment, deep, 309
dorsum, contusions, 415
fractures, 512–4
astragalus, 513
mid-tarsal region, 513
os calcis, 513–4
injuries in cricket, 169, 171
in ski-ing, 255
strain, acute, 443
Football, achilles tendon tear, 137
concussion in, 139
injuries, 137–41
collision, 141
muscle, 141
types, 137
myositis ossificans, 137
rugby, *see* Rugby football
training, second week, 85
Force(s), acting on spine, 567–9, 571–2
of blades in fencing, 179, 183–8
injuries due to reversal, in fencing, 177
Forearm, contusion of tissue, treatment, 370
in baseball, 152
Fractures, associated with main vessel injury, 352
and avascular bone necrosis, 490–2
in basketball, 156
bone absorption at surfaces, 489–90
bone fragments, angulation, 496
blood supply, 477
overlapping, 494–5
relationship, between, 494–9
rotation, 496–8
bony union, 493–4

Fractures—cont.
in boxing, 164–5
prevention, 165
changes, associated with, 485–92
generalised, following, 489
chip, of lower limbs, 414
conditioning by exercise, 91–2
and damage to blood vessels, 487
effects on subcutaneous tissue, 486
effect on tendons, 487
factors influencing healing, blood supply, 476–9
bony fragments, 477
factors which determine subsequent function, 493–500
'fatigue', *see* Fractures 'stress' or 'fatigue'
healing, 474–92
factors influencing, 476–85
age, 484–5
bone fragments, movement between, 482
deficiency diseases, 485
infection, 482–4
natural, processes, 474–6
relationship between bone ends, 479–82
special problems in athletes, 510
time factor, 519
and Volkmann's Ischaemic Contracture, 487
immobilisation, traction, 504–6
importance of treating soft tissues, 450
incidence of emboli, 488
involving joints, 489
treatment, 330
involving muscle, 486–7
involving peripheral nerves, 488–9
involving soft tissues, 499–500
involving veins, 488
'march', *see* Fractures, 'stress' or 'fatigue'
in motor racing accidents, 224
and muscle death, 487
and muscle wasting, 499–500
and myositis ossificans, 486–7
oedema following, 486
production, 468–74
by direct violence, 469–72
by force transmitted through muscle or tendon, 473
by indirect violence, 471–2
'stress' or 'fatigue', 473
by transmitted force, 472–3
results of delay in treatment, 489–90
in rugby football, 231
'ski-pole', 252, 256
skin changes in, 486
'stress' or 'fatigue', 533–49
causes, 473, 533–5
history and nature, 533
incidence, 535–7
mechanism of production, 535
nature, 533–5
treatment, 537–49
for treatment of individual bones *see* names of bones
treatment, immobilization, 502–6
fixation, external, 503–4
internal, 506
objectives, 493
principles, 493–511
preservation of function, 506–9
reduction, 500–2
manipulation, 501
open, 502
traction, continuous, 502
time factor, 510
in water ski-ing, 247, 248
see also bones, joints and regions involved, and individual sports
Fracture-dislocations, spinal, 587–92
dorsal or lumbar region, 589–90
Fulcrum, of spine, action in movement, 73–6
movement and mechanical forces, 568–9
see also Posture, Movement
Galvanism, anodal, 308
for bursal injuries, 331
interrupted, 307–8
Game, shooting, injuries, 191–9
Gibney boot, 157
Girdlestone's operation for osteoarthritis, 466
Gloves, for wicket keeping, 170

Glucose, supplements before and during events, 128–9
Goalkeepers, football, injuries to, 139
'Golfers' elbow', 202, 205, 216
  injuries, 200–5
  low-back disorders, 134
  muscle action, 76
  professional and amateur, difference in injury, 204
  training, 203
Grafts, of divided blood vessels, 347–8
  for loss of whole skin thickness, 457
Gravity, effect on spine, 567–8
Guns, 'burst' injuries from, 195
  fitting, 196, 199
  handling, 196–9
  'headache', 194
  precautions, 196–9
  and prevention of accidents, 192
  recoil injuries, 193–5
Gun-shot wounds, accidental, 191–2

Haemangiomata, 354–5
Haematoma(s), bone and cartilage, treatment, 331–2
  and buccal varidase, 165
  in boxing (black eyes), 161
  complications, 278–81
  deep-seated, treatment, 280
  formation, over fascial planes, 263
  in fractures, 474
    formation, 474
  pulsating, 352–3
  result of direct trauma, 276
  treatment, 319, 320–2
    injection therapy, 314
  unabsorbed, 280, 456
  see also Contusions
Haemoglobin, increase during training, 45
Haemorrhage, acute, localized, intramuscular, in baseball, 152
  intracranial, in boxing, 164
Hands, blisters, in rowing, 227
  circulation, 359
  contused, deep x-ray treatment, 309
  dorsum, contusion sprains, severe, treatment, 365
  contusion sprains, treatment, 364–5
  wicket keepers', 171
  see also Fingers, Thumbs
Hats, riding, design, 213, 214
  see also Helmets
Head injuries, 405–6
  in baseball, 153
    prevention, 153
  in jockeys, 212
  in motor racing, 224–5
  in rugby football, 231
  in ski-ing, 251–2
  in water ski-ing, 248
Headache, gun causing, 194
Healing of injuries, phases, negative and positive, 291
  see also Fractures
Heart, athletic and average, differences, 40
  damaged, in rowing, 226
  disease, occurrence among athletes, 116
  effect of training, 40
  and lung, effects in maximum performance, 53–5
  'strain', in women athletes, 117–8
  training, 42–5
  see also Cardiac
Heat, acclimatisation, 47–9
  effect on tissues, 285
  elimination by body, 47
  see also Acclimatisation, Temperature, Weather
Heels, blistered, in rowing, 228
Helmets, crash, compulsory in motor racing, 224
  see also Hats
Heparin, 354, 355, 356
Hockey, injuries, 206–9
  precautions, 207
Honey, before and during events, 128–9
Horses, kicking, falling, or rolling on riders, injuries, 210, 211–2, 214
Humerus, fractures, in baseball, 152
    stress, 548
    supra-condylar region, 529–30
    treatment, 530
Hyaluronidase, 332

Hyaluronidase—cont.
in athletic injuries, 92
injection therapy, 313
Hydrocortisone, in athletic injuries, 92
in clavicle injury, 402
injection therapy, 313
scapula injury, baseball, 148
shoulder joint disorders, 400
treatment of tennis elbow, 219
Hypertension, and the women athlete, 116

Ibbotson, D., and training for peak performance, 85
Immobilization of fractures, 502–6
Impact, injury due to, in fencing, 178
Impulses, nerve, 20
Incisions, treatment, 319
Index, Ponderal, 94
Infection, influence on fracture healing, 482–4
Injection therapy, 312–5
aims, 312–3
analgesics in, 315
combined with physiotherapy, 313
solutions used, 313
technique, 313–5
Injury, injuries, aggravating existing conditions, 467
arterial, treatment, 339–58
basketball, 155–8
'burst', from shotguns, 195
causes, 274–5
classification and treatment, 318–58
complications following, 455–67
classification, 456
difficult cases to make symptom free quickly, 453–4
direct and indirect, 134
and early return to sport, 289–90
according to time in season of sport, 293
factors influencing, 278–87
fatal, in rugby football, 231
in golf, 200–5
history, 266–8
in hockey, 206–9
importance of prevention, 136
to jockeys, analysed, 211
mechanism of production, 133–6
long continued stresses, 135–6
outside agency, 133–4
self-produced, 134
in motor racing, 224–5
muscle, treatment, 320–7
see also Muscles
nerve, 332–9
see also Nerves
overload, in baseball, 151–2
pain in, 315–6
recoil, 193–5
risk, 133
in rowing, 226–9
in rugby football, analysed, 230
spinal, 575–92
in swimming, 236–41
as tactical weapon, in cricket, 134
tendons, treatment, 320–7
treating difficult cases, 445–54
treatment and classification, 318–58
'treat and train', 91
trivial, neglect, 139
in water ski-ing, 242
in winter sports, 250–60
see also names of individual sports
Innervation ratio, 16
Intensity Duration Curve, 271
in never injury, 334–65
International Water Ski Union, 242
Interval methods of training, 87
Ischaemia, signs and treatment, 351–2

Javelin thrower's arm or elbow, 381–2
Jaw, fractures in boxing, 164
see also Maxillo-facial injuries
Jockeys, fitness in, 213
injuries to, 210–13
steeplechase, clavical fracture and return to racing, 79, 92
shoulder dislocations, treatment, 394
Joints, general
cartilaginous, 7
fibrous, 6–7
injuries, classification and treatment, 327–30

Joints—cont.
diagnosis, 263–74
dislocations, treatment, 329–30
in football, 138–9
fractures, treatment, 330
internal derangement, treatment, 328–9
treatment and classification, 327–30
involuntary movement, 302
involvement, examination, 270
in fractures, 489
locking, and diagnosis of injury, 267
loose, causes, 459
operative treatment, 459–60
lubrication, 265–6
movement, mechanics, 77–8
range of movement, 10
sprains, treatment, 327
stiff, complication of injury, 457–9
synovial, 7–10
ball and socket, 7, 9
condyloid, 9
hinge, 9
pivot, 9
plane, 9
saddle-shaped, 9
types, 7–8
types, 6–10
unstable, causes, 459
Joints, named
acromio-clavicular, 402–3
injuries, treatment, 402–3
subluxation, 402
ankle, examination under anaesthesia, 272
fractures, 514–7
classification, 516
compression, 516–7
in football, 138
tibio-fibular diastasis, 517
injuries, 438–43
in baseball, 152
in ski-ing, 253
manipulation, *see* Manipulation
protection, in basketball, 157
sprains, in basketball, 155
in football, 138–9
carpo-metacarpal of thumb, fracture in boxing, 164
elbow, dislocation, 379–81
fractures involving, 529
injuries in baseball, 149–50
Javelin thrower's, 381–2
'pulled' and 'pushed', 379
in young pitchers, 153–4
see also Tennis elbow, Golfers' elbow
finger, dislocations, in baseball, 152
foot, sprains, 443
hip, injuries, in water ski-ing, 247
Joint, interphalangeal, button-holing, 362
sprains, treatment, 360–1
stiff, treatment, 361–2
Joints, intervertebral and spinal movements, 563
strains, 582
knee, aspiration of synovial fluid, 295
injuries, 420–30
abduction, in football, 138–9
baseball, 152
in basketball
Compton's, 171, 172
diagnosis, 423–5
direct, 425–6
flexion, in football, 138–9
indirect, 426
operative treatment, 432
in ski-ing, 252–4
synovitis, traumatic, treatment, 426–8
in water ski-ing, 247
ligaments, anatomy, 422–3
manipulation, *see* Manipulation
movements, 420–1
relationship of ligaments and meniscii, 421
muscles acting on, 423
prevention of injury, 423
rotation injuries in football, 138–9
strains, in golf, 202
triad, treatment, 430–5
lumbar, strains, in golf, 201
metacarpo-phlangeal, dislocation, in baseball, 152
fractures in boxing, 164

Joints—cont.
sprains, treatment, 360
worsening during treatment, 445
shoulder, anatomy, applied, 385
bursitis, subacromial treatment, 390–1
calcification, 389
capsulitis, cause, 385–6
treatment, 395–7, 399–402
deformity, in professional golfers, 204–5
dislocations, harness for, in fencing, 177
frozen, 398–402
symptoms, 398–9
treatment, 399–402
injuries, 383–402
activity, 387
bruising, and gun recoil, 194
classification, 387
in cricket, 170
diagnosis, 387–8
injuries, dislocation, 393–5
periarthritis, 395–8
in fencing, 177
to jockeys, 212
in rugby football, 231, 232
in ski-ing, 252
sprain, 388
subluxation, 392–3
in water ski-ing, 247
manipulation, 400–1
movements, types, 386
'swimmers', 236–8
see also Muscles, biceps, long head
spinal, 'sprains' and 'strains', 579–82
'strains', 579–82
terminology of disorders, 579
see also Column, spinal
temporo-mandibular, lesions, 405–6
wrist, fractures, 525–6
manipulation, 302, 303, 304
sprains, treatment, 366–9
tenovaginitis, treatment, 369–70

Kidneys, complication in sport injuries, 357–8
Knee jerk, mechanism, 22
joint, *see* Joints, knee
Kohn, F. Blankers, 121
Kuts, V., and training for peak performance, 85

Laboratory tests, in diagnosis of injury, 273
Labyrinth, bony, of inner ear, and balance, 24–7
Lacerations, in boxing, treatment, 160–1
Lactic acid, concentration, 41, 53, 54, 56–7
injections, 313
Landy, John, and training for peak performance, 85
Leg, fractures, in football, 139
in rugby football, 234
injuries, in rugby football, 231
in ski-ing, 259
in swimming, 238
lower, injuries, in ski-ing, 253
stability, in diagnosis of injury, 268
see also Joints, knee, ankle, Femur, Limb(s) etc.
Leukotaxine, action on leucocytes, 281
Lever action of muscles, 73–6
Ligaments, cruciate, injuries, in basketball, 156
damages, in basketball, 156
and extent of movement, 10
fibulo-calcaneal, injuries in basketball, 156
injuries, in ski-ing, 253–4
interspinous, damage, 579
of knee joint, anatomy, 422–3
injury, knee joint triad, 430–5
lateral, injuries, 434
strain, in golf, 202–3
medial, injuries, 428–9
sprain, in football, 138
strains, in golf, 202
tears, 431, 432
spinal, damage, 578–9
structure, 556–7
stretching of, difficult to make symptom free quickly, 454
supraspinous, damage, 579
Limb(s), fractures in boxing, 165

Limb(s)—cont.
injury, treatment as whole, 359
lower, injuries, 414–444
movements, *see* Movements
see also Arms, Legs
Loading, of guns, 198–9
Lovelock, Dr., and training for peak performance, 85
Lubrication of joints, 265–6
Lunge, in fencing, description, 175
Lungs, and alveolar capillary function, 38
function, bellows, and maximum breathing capacity, 37–8
and heart, effects in maximum performance, 53–5

McMurray's displacement osteotomy of femur, in osteoarthritis, 466
rotation tests of knee, 424, 431
Malleoli, contusions, 415
Manipulation, of adhesions, under anaesthesia, 272, 305
after injury, critical survey, 450–1
of ankle and foot, 436, 440, 441
for chronic traumatic myositis, 460–1
of fractures, 501
gentleness in, 305–6
for joint stiffness, 458
of knee joint, 431, 432, 433
lower cervical and cervico-dorsal junction, 408
of menisci, 430
method, 303–4
of muscle, 305–6
of naviculo-cuneiform-metatarsal joint, 442
principles, 301–3
of 'pulled' and 'pushed' elbow, 379
shoulder joint, 400–1
spinal, 409, 410
under anaesthesia, 272, 304–5
upper limb, 383, 384, 386, 389
Marathon, effect of sun during, 147
unconsciousness during, 33
Mask, in fencing, 178
Massage, after injury, 450
in bursal injuries, 331
general application, 301–2
and injury, 84
objects, 301
Maxillo-facial injuries, in baseball, 153
prevention, 153
see also Jaw
Meal(s), before fight, 167
'Pregame', 129
regular hours during training, 127
Meniscus, injuries, differential diagnosis, 424
lesions and injury, 429–30
torn, in football, 138
Menstruation, effect of sports on, 118–9
games-playing during, 118
interruption, 120
use of tampons, 119
Mental approach of athlete, *see* Psychological factors
Mental health, and the athlete, 111–12
Mesomorphy, 94–104
definition, 94
dominance in sport, 99
'somatotonic' personality, 99
Mesopene (fat-thin man), 97
Metabolism, anaerobic, 41
Metacarpal bones, fractures, 524–5
in boxing, 164
reduction, 525
varieties, 524
traction, 525
Metatarsal bone, bruising or fatigue fracture in bowlers, 170, 172
fractures, stress, clinical findings, 538
nature, 537
prognosis, 539
site, 537
treatment, 538–9
fifth, injury, 134
Minerals, in the diet, 126
Motor racing accidents, 224–9
Mountaineers, maximum breathing capacity, 37
Movement(s), axis, of spinal column, 563–6
body, and spine, 569–70
complicated, mechanism, 27–32
training, 28–31
in diagnosis of injury, 267

Movement(s)—cont.
in fencing, 173–7
integrated, and posture, 80
joint, mechanics, 77–8
limb, and body structure, 1–32
lever action of muscles, 14–15
and ligaments, 10
and nerves, 16–22
range, and joints, 10
range, limiting factors, 10
strength, and muscles, 14
and lumbar intervertebral discs, 594–5
mechanics, 73–6
in fencing, 175
and posture, 62–81
muscular, and proprioceptive neuromuscular facilitation, 310–12
spinal, 561–70
in sport, applied anatomy, 78–9
Muscle(s), general
acting on spine, 567–8
action, 76–7
activator group, 77
antagonists, 77
in golf, 76
in tennis, 77
actions of nerves on, 22–4
architecture, 13–14
attachments, 14
classes, 10
contusion ('Charlie horse'), in basketball, 156
cramp, treatment, 89
extensibility and flexibility, and diagnosis of injury, 263–4
increasing strength by training, 88
injuries, in cricket, 168
in diving, 240
in football, 137–8, 141
by indirect trauma, 276–7
prevention, 137
in rugby football, 235
treatment, 320–7
in water ski-ing, 247
involvement, examination, 269
in fractures, 486–7
manipulation, 306–7
over-use, 326
partial ruptures, treatment, 323–4
pulled, in fencing, 177
in hockey, 207
prevention, 142–4
in runners, 142–4
cause and prevention, 145–6
rupture, complete, treatment, 324–5
strains and bad posture, 69
in golf, 202
in rowing, 226–7
in swimming,
treatment, 322
and strength of movement, 14
strength, and training, 34–6
tears, difficult to make symptom free quickly, 453
and fast bowling, 134
repair, and return to activity, 90–1
and tennis elbow, 217–8
treatment and healing, 577–8
wasting, treatment, by interrupted galvanism, 307–8
by surging faradism, 308
Muscles, named,
abdominal, strained, in rowing, 226–7
anterior tibial, contusions, 417–8
biceps, long head, lesions, 391
predisposing factors to injury, 391–2
rupture, treatment, 382
buttock, strains, 418
calf, contusions, 418
strains, 435–8
tears, 435–8
gluteal, contusions, 416
hamstring, strains, 419
torn, in cricket, 168, 293
iliopsoas, strain, 418
infraspinatus, in baseball, 148
intercostal, contusions, 406
peroneus brevis, and injury of fifth metatarsal, 134
quadriceps, action on knee joint, 423
contusions, 416–7
injury in football, 137
loss of extensibility, treatment, 417
strains, 419

Muscles—cont.
skeletal, activity, continuation, and blood supply, 16
antagonists, 15
attachments, 11–13
bipennate, 13
contractions, 16
intermittent, 16
lever action, 14–15
prime movers, 15
structure, 10–11
synergists, 15
unipennate, 13
voluntary, 11, 12
spinal, injuries, 575–8
site, 576
symptoms, 576–7
treatment, 577–8
posterior, contusions, 410
strains and tears, 412
structure, 559–60
striated, *see* Muscles, voluntary
supraspinatis, fraying or rupture in baseball, 148–9
thigh, strains, 419
strain in bowling, 170, 172
triceps, injury, in Baseball, 151–2
voluntary, types of fibres, 79–80
see also Contractions, Volkmann's Ischaemic Contracture
Muscle-tendon-bone unit, acute and chronic overload, 151
injuries in basketball, 155
Myelograms, 271
Myositis, chronic traumatic, treatment, 460–1
ossificans, 322
complication of injury, 137, 461
and fractures, 486–7
occurring in immobilised joint, 416–7

Nasal bones, fractures, in boxing, 159–60
in cricket, 169
National Water Ski Tournament, first, 242
Neck, 'broken', 583
injuries, in ski-ing, 251–2
see also Spine
Necrosis, bone, avascular, and fractures, 490–2
fat, acute, 434
Nerve(s), actions on muscles, 22–4
cell, 18
circumflex, vulnerability, injuries to, 385
conduction test, in nerve injury, 334–5
fibre, structure, 16–20
impulses, 20
injuries, 332–9
electrodiagnosis, 333–7
electromyography in, 335–6
examination for, 270
investigations, 333
prognosis, 339
treatment, 337–9
of closed injuries, 337–8
open wounds, 338–9
involvement in pain, 315
peripheral, involved in fracture injuries, 488–89
spinal, effects of dislocations and fracture-dislocations on, 587–8
Neurapraxia, 337
prognosis, 339
Neurone, *see* Nerve cell
Neurotmesis, 336
prognosis, 339
Norwegian 'Fartlek Interval Training', 87
Nose bleeding, *see* Epistaxis
Nose, *see* Nasal bones
Nutrition and the athlete, 123–9
Nutrition, historical factors, 123
Nutrition, personal factor, 124
see also Diet

Oedema, associated with fractures, 486
localized, in physical treatment, 283–5
Olecranon fractures, in baseball, 149
Operative treatment, critical survey, 452–4
definite indications for, 453
see also specific injuries and regions

Organs of balance, 24
Os calcis, fractures, 513–4
stress, clinical findings, 539–40
prognosis, 540
treatment, 540
Ossicles, elbow, in baseball players, 150
Osteoarthritis, course, 463–4
deep x-ray therapy for, 309
of fingers in spin bowlers, 170
following inactive slumping position, 69
treatment, 464–7
general, 465
injections, 466
local, 465–7
operative, major, 466–7
minor, 466
physiotherapy, 465
preventive, 464–5
Osteochondritis, elbow, in baseball, 149
Osteochondritis, of knee joint, 434
Osteochondritis dissecans, after injury, 462
Osteotomy, femur, in osteoarthritis, 466
Otitis media, in water skiers, 247
Overweight, considerations, 127
Oxygen, breathing, effects, 55–7
during exercise, 56
effect on recovery, 56–7
exercise before and after, 56
lack, limiting factor in performance, 88
tension, arterial blood, effect of heavy exercise upon, 54
transport, 36–46
limiting factor in maximum performance, 53–5

Padding, in football, 139, 140
Pain, analgesics for, 316
back, low, and draughts, 285
and inactive slumping posture, 69–70
and lumbar disc lesions, 600–2
causes, 315
in diagnosis of injury, 266–7
in injury, 315–6
and old injury, 316
Parnell's simplified somatotyping, 97
Parry, in fencing, description, 175
seconde, in fencing, 175
Parturition, in athletes, 121
Patella, contusions, 415
direct injury, 425–6
fractures, 519–20
stress, clinical findings, 544
site, 544
treatment, 544–5
treatment, 520
function, 519–20
see also Joints, knee
Pathology of sports injuries, 274–87
Pelvic ring, fractures, 522–3
Pelvis, examination in women athletes, 116
female, athletic, and pregnancy, 120–1
fractures, 522
in jockeys, 212
Penetration, injuries due to, in fencing, 178
Performance, maximum, factors limiting, 53–9
Periarthritis, 395–8
of the shoulder, differential diagnosis, 397–8
treatment, 395–7
Personality, 'cerebrotonic', 98
'somatotonic', 99
'viscerotonic', 98
Phalanges, fractures, 524
distal, fractures, in catchers, 152
Phlebectasia, 355
Physiological and psychlogical approach to athletics, 105–12
Physiologist and athlete, different approaches to training, 88
Physiology, and stress, 33
sub-division of athletic events, 33–4
Physiotherapy, 91, 92
for bursal injuries, 331
in clavicle injury, 402
combined with injection therapy, 313
and localized oedema, 283–5
in 'pulled' and 'pushed' elbow, 379
shoulder joint, 385, *et seq.*

Physiotherapy—cont.
in sprains of wrist, 368
see also individual headings and references to treatment throughout
Plantar fasciitis, 443
Plaster, adhesive, strapping, uses, 300–301
casts, contraction of muscles in, 289
Plaster-of-Paris, support, 299
use for external fixation of fractures, 504
Plastic supports, 299
Players, injured, and return to play, 233
see also individual sports
Position, body, perception, 27
Posture, active alerted, 65–6, 68–9
body position, 71–2
balance, 24–7
and changes in spinal column, 571
definition, 62
during training, 85
female, improvement, 114–15
five fixing levels, 62–3
in golf, 203, 204
and gravity, 63–4
inactive slumping, body positions, 69
harmful effects, 69–71
influence on athletic injury, 278
and integrated movement, 80
and limb movements, 22–7
and mechanics of movement, 62–81
passive supported, 65–68
phases, 62
and physical well-being, 561
slumping, dangers, 64–5
spinal, development, 574
spine in, 570–4
standing, 22
stretch reflex, 22–3
see also Stance
Potassium balance, 349
causing death in late vessel repair, 352
Prawns, and force of gravity experiments, 24–5
Pregnancy, continuation of sport after, 121
and sport, 120–1
Prevention, of accidents, in Rugby football, 232
with guns, 192
riding, 213–4
of injuries, 136
in basketball, 157–8
in boxing, 165
in rugby football, 235
muscular, 137
of osteoarthritis, 464–5
of tennis elbow, 219–20
and treatment, 294
Procaine, in injection therapy, 313
Processes, articular, fractures, 584
spinous, fractures, 583
transverse, fractures, 584
Progress, indication, 298
Proprioceptive neuromuscular facilitation, 310–12
Protection, in rugby football, 232
Protector, aluminium, for scrotal area, for batting and wicket keeping, 169
Protein, amount required during training, 126
calorie value, 125
foods high in, 125
need, 125
Pruritis, occurrence in women athletes, 116
Psychological factors, in athletics, 105–12
before events, 89
see also Mental health, Personality, Stress
Pseudarthrosis, Batchelor's, in osteoarthritis, 466
Psychiatrist, need for, 109–10
Pubis, fractures, stress, clinical findings, 547
site, 547
treatment, 547
Pulse, athletic, 41–2
frequency, and energy expenditure, 43
recovery, 44
Punch-drunk syndrome, in amateur boxers, 162

Qualities, in athletes, 106

Radiation, ultrasonic, treatment, 309
Radiographs, in diagnosis of injury, 270–1
Radius, fractures, 528
lower end, 527
and ulna, 529
Raynaud's phenomenon, 357
Reaction, quick, 108
Recoil, injuries, 193–5
cure, 194–5
Recovery, phases, negative and positive, 291–2
Reduction of fractures, 500–2
open, 502
Reflex arc, of nerves, 20–21
stretch, 22–3
Rehabilitation centres, after injury, 449
Rehabilitation by trainer, 297–8
Relaxation, before an event, 106
Renal complications, in sporting injuries, 357–8
Renal units, 341
Repetition in training, 108
Respiration, apparatus, 37–40
control, 38–40
mechanism, during exercise, 54–5
see also Lungs
Rest, and exercise, in treatment, 288
after injury, 448–9
Retina, detached, in boxing, 164
Ribs, fractures, in jockeys, 212
stress, of first, 548–9
clinical findings, 549
site, 549
treatment, 549
treatment, 531
injuries to, in boxing, 167
Riding accidents, 210–15
prevention, 213–4, 215
Rope, tow, injuries, in water ski-ing, 247
Rotation tests of knee, McMurray's, 424, 431
Rowing, injuries in, 226–9
Rugby football, injuries in, 230–5
in relation to positions on field, 231
prevention, 235
suggested measures, 232
and return to play, 232, 233
Runners, blisters, 147
long distance, and pulse recovery, 44
middle distance, maximum breathing capacity, 37
oxygen transport, 36
pulse, 42
ventilation equivalent, 40
'pulled' muscles, 142–4
in, cause and prevention, 145–6
somatotyping, 100, 101
and spiking, 147
stiffness in, dangers, 145–6
see also Athletes
Running, cross country, calorie expenditure, 126

Saccule, 24
Sacrum, injuries, 411
Salicylic acid-urea solution, in injection therapy, 313
Salt (sodium chloride) daily intake, 127
necessity, 126
Scalp, contusions, 405
Scaphoid, carpal, fractures, 526–7
Scapula, fractures, 530–1
fractures, treatment, 531
injuries in baseball, 148
Scar, contracted, treatment, 457
Sciatica, and lumbar disc lesions, 603
Semilunar bone, dislocations, 365–6
Sepsis, contributory factor to 'pulled' muscles, 144
superficial, complication of injury, 456
Septum, deflected, in boxing, 159–60
Sheldon's somatotype classification, 94, 97, 98
Shin, soreness, in runners, dangers, 145–6
Shoe, running, design, 144
Short wave treatment, after injury, 452
Shooting, game, injuries, 191–9
Shoulder, *see* Joints, shoulder
Sinusitis, in swimming, 241
in water skiers, 247
Ski-ing, snow, injuries, distribution, 251, 260
lack of training, 259

Ski-ing—cont.
mechanism of, 255–7
types, 251
water, *see* Water ski-ing
Skin, injuries to, management, 318–20
changes and fractures, 486
grafts, 457
Skull, fractures in boxing, 164
Sleep, requirement, 86
Slumping, *see* Posture
Smoking, and intermittent claudication, 357
and maximum breathing capacity, 38
Somatocharts, 95, *et seq.*
Somatotypes, distribution among athletes, 100–1
significance in sport and athletics, 94–104
and injuries, 104
variations from, in choice of sport, 94, 102
Soreness, in rowing, 229
Spasm, arterial, and fractures, 487
treatment, 348
Spiking, during running, 147
in baseball, 153
Spine, *see* Column, spinal
Splints, plastic, 299
used for fractures, 504
Spondylosis, following inactive slumping position, 69
Sport, importance of, 105–6
movement in, applied anatomy, 78–9
women in, 113–22
see also individual sports and injuries
Sprains, ankle joint, 438–43
in basketball, 155
classification, 439
causes, 438–9
in football, 138
mild, 439–40
moderate, 440
treatment, 441–3
severe, 440–3
treatment, 441–3
in boxing, 164–5
of foot joints, 443
joints, severe, treatment, 328
treatment, 91, 327–8
ligamentous, treatment, 327
moderate, treatment, 327–8
shoulder joint, symptoms and treatment, 388
Sprinters, born, not made, 107
and breathlessness after race, 16
somatotyping, 100, 101
see also Athletes, Runners
Spurs, elbow, in baseball, 150
Squash rackets, detached retina in players, 164
Stability, and diagnosis of injury, 268
Staleness, signs and symptoms, 111
Stance, in fencing, 173
see also Posture
Standing, posture, 22
Statocyst, of prawn and force of gravity, 24–25
Steeplechasing, accidents, 213
see also Riding accidents
Sternum, fractures, 531
Stiffness, complication of injury, treatment, 457–9
after exercise, 84
dangers, in runners, 145–6
and diagnosis of injury, 268
Stitch, treatment, 89–90
Strains, acute arch, in baseball, 152
of calf muscles, 435–8
of foot, acute, 443
and inactive slumping position, 69
intercostal muscles, 408–10
lumbo-sacral, 581–2
treatment, 582
of muscles of lower limbs, 418–20
treatment, 322
occupational, influence on athletic injury, 278
postural, influence on athletic injury, 278
sacro-iliac, 580–1
treatment, 581
tendon and ligament, vulnerability of women, 115–6
Strapping adhesive, or elastoplast, uses, 300–01
Strawberries, *see* Abrasions

Stresses, of blades in fencing, 183
long-continued, 135–6
athletes, 110–11
signs and symptoms, 111
see also Psychological factors
Structure, body, bones, 3–4
joints, 4–10
and limb movements, 1–32
muscles, 10–16
nerves, 16–22
variation, 1
Subluxation, acromio-clavicular, joint, 402
of cervical spine, 590–1
of shoulder joint, 392–3
sterno-clavicular, treatment, 403
Sudeck's atrophy, 492
SUELMEX, 153
Sugar, supplements, before and during events, 128
Sun, effect on marathon, 147
Support to damaged parts, 229–301
lumbar, in car and aircraft, 203
Supraspinatus strain, tear or rupture, cause and treatment, 389–90
syndrome, in fencing
Surgery, manipulative, *see* Manipulation
Sweat, and intake of salt, 126, 127
Sweating, from hands and feet, treatment, 357
Swelling, antecubital, in baseball, 150
in diagnosis of injury, 268
traumatic, and anti-inflammatory enzymes, 316
see also Oedema
Swimmers, body temperature, 50
Swimming, calorie expenditure, 126
injuries in, 236–41
breaststroke, 238, 239
crawl stroke, 236–8
turning, 240
phases of training, 87
Sympathectomy, for hyperidrosis, 357
in intermittent claudication, 356
Symptoms, of staleness, 111
of stress, 111
Synapse, 21
Synovitis, traumatic, of knee, treatment, 426–8

Tears, *see* Muscles, Tissue, etc.
Teeth, damage, in wicket keeping, 169
Temperature, body, control, 47–53
control, heat acclimatisation, 47–9
normal mechanism, 47
rise during exercise, 50–2
and diagnosis of injury, 268
see also Heat, Cold, Weather
Tenderness, areas of, 285–6
and diagnosis of injury, 269
Tendon, Achilles, injury in cricket, 168
in hockey, 208
in ski-ing, 254–5
rupture, 436–8
tear, in football, 137
teno-synovitis, in runners, 144
prevention, 144
adductor longus, and myosistis ossificans, 137
associated with fractures, 487
avulsion, treatment, 325–6
complete rupture, treatment, 324–5
injuries, treatment, 320–7
plantaris, rupture, in baseball, 152
popliteus, injury, 435
soreness, prevention, 146
see also Muscle-tendon-bone unit
Tendonitis, bicipital, in baseball, 149
suprasinatus, in swimming, 238
in bowling shoulder, 170
treatment, 326
Tennis, calorie expenditure, 126
importance of warm-up, 219–20
muscle action, 77
Tennis elbow, 216–23
causes, 216–8, 371–2
and change in 'habit' performance, 220–1, 223
definition, 216
and early ball play, 223
in fencing, 177
in golfers, 202, 205
operative treatment, 378
pathology, 281

Tennis elbow—cont.
personal experience of Dan Maskell, 221–3
points of tenderness, 372–3
prevention, 219–20
six different types, 373–8
and slumping posture, 70
symptoms, 372
treatment, 218–9, 223, 374, 375, 377, 378
Teno-synovitis of the Achilles tendon, in runners, 144
prevention, 144
chronic traumatic, treatment, 461–2
in rowing, 227
Thoracic outlet syndrome, associated with inactive slumping posture, 70–71
Tenovaginitis, De Quervain's, 369
stenosing, difficult to make symptom free quickly, 453
early diagnosis, 269
treatment, 326, 369–70
see also Finger, trigger
Therapy, *see* Treatment
Thigh, fractures, in ski-ing, 252
Thoracic wall, contusions, 406
Thorax, fractures, 531
Thromboangiitis obliterans, 357
Thrombo-phlebitis, complication of injury, 462
superficial, 356
Thrombosis, arterial, 354
complication of injury, 455
effort, 353–5
venous deep, treatment, 355–6
iliac, femoral and subclavian, 354
Throwing, and strain on brachialis muscle, 170
Thumbs, damage to during wicket keeping, 170
fractures in boxing, 164
injuries in baseball, 152
trigger injuries, 194
Tibia, fractures, 518
stress, clinical findings, 542
site, 541–2
treatment, 543
rotation, extension and flexion on femur, 420–1
subcutaneous border, contusions, 415
Tibia and fibula, fractures, 518–9
stress, clinical findings, 543
site, 543
treatment, 543–4
treatment, 518–9
Tibio-fibular diastasis, 517
Time factor in fracture healing, 510, 519
lag of repair of injury, 352
Tinea pedis, in swimmers, 241
Tiredness, and tennis elbow, 218
Tissues, cooling and heating, effects, 285
fibrosis, 281–2
fibrous, pathology, 282–5
heating and cooling, effects, 285
soft, injuries, diagnosis, 263–74
tears, repair, 275–6
Toes, contusions, 415–6
fractures, 512–3
injuries in cricket, 169, 171
Traction, continuous, in reduction of fractures, 502
Trainer, rehabilitation by, 297–8
Training, complicated muscle movements, ability, 28–31
diet during, 126–8
effect on circulation, 45
effect on heart, 40, 42–5
and fitness, 82–93
general, 83–7
circuit, 87
graduated exercise, 83–5
Norwegian 'Fartlek' Interval Training, 87
postural, 85
specific exercises, 85–7
weight-lifting for athletics, 86–7
weight reduction, 83
general, walking and running, 83–5
for golf, 203
heart and circulation, 42–5
for international competition, 89
light and heavy work combined, 103
mileage covered by marathon runner, 145
and muscle strength, 34–6

Training—cont.
retraining, 35–6
repetition, 108
in rugby football, 231
for the special event, 87–90
'velocity', 103
Transport, air, cost, 358
Trauma, direct, pathology, 276
indirect, pathology, 276–7
Treatment, basis, 288–91
by doctor and specialist, general principles, 295–6
electrical, *see* Faradism, Diathermy, etc.
first aid, principles, 294
fundamentals, 292
general, 288–317
home, 297
indication of progress, 298
injection, 312–5
interferential, 310
local, 296
manipulation, 302–7
massage, 301–2
physiotherapy, general principles, 296–7
planning, 293
preventive, 294
proprioceptive neuromuscular facilitation, 310–12
'purposeful aggravation', 298
rehabilitation by trainer, 297
short-wave diathermy, 307–9
support to damaged part, 299–301
ultrasonic radiation, 309
x-ray, deep, 309
Triad, O'Donohue's, 156
Triceps, *see* Muscles, triceps
Trichomonas vaginitis, occurrence, 116
Trimalleolar bone, fractures, in jockeys, 212
Trochanter, great, contusions, 414–5
Trunk, injuries, 406–12
Tuberosity, ischial, contusions, 415

Ulna, fractures, treatment, 528–9
stress, clinical findings, 548
site, 548
treatment, 548
styloid process, fractures, treatment, 527
see also Radius and ulna
Ultrasonic radiation treatment, 309, 452
Unconsciousness, in boxers, treatment, 161–2
during marathon, 33
Uric acid, plasma, high and calcified deposits in shoulder joints, 390
Utricle, 24

Vaginal discharge, occurrence, 116
Varidase, buccal, administration, oral, 316, 332
Vascular injury, *see* Blood vessels
Vascular surgery, 339–58
Veins, associated with fractures, 488
ligation, 348–50
phlebectatic, 355
treatment, 350–1
treatment, 341–2
varicose, and inactive slumping position, 69
treatment, 355
Ventilation equivalent for oxygen, 39–40
Vertebrae, bony, structure, 551–2
cervical, articulation, 566–7
fractures of bodies, 584–7
clinical findings, 586
site, 584–6
treatment, 586–7
movements between, 563–6
see also Articulations, intervertebral
Vitamin B group, requirement in the diet, 126
Vitamins, in the diet, 126
Volkmann's Ischaemic contracture, complication of injury, 455–6
associated with fractures, 487
Vulvo-vaginitis, monilial, occurrence, 116

Walking, as exercise, 83–4
Wall, thoracic, contusions, 406–8
Warm-up, before rugby football, 233
importance in tennis, 219–20
justification, 52–3
and prevention of pulled muscles, 142

Water polo, injuries in, 241
Water ski-ing, beginners' lessons, 244–6
dangers to untrained participants, 244
description, 246
drownings, 248
equipment, 246
falling position, 247
injuries in, 242
causes, 246–8
suggested regulations, 248–9
types, 246
Weather, cold, and pulled muscles, 146
sun, and athletics, 147
and tennis elbow, 217
see also Cold Heat, Temperature
Webbing and felt supports, 300
Weight, reduction, in training, 83
Weight lifters, and pulse recovery, 44
Weight-training for athletics, 86–7
type training, 102–3
after injury, critical survey, 446–8
graded exercises, 447–8
'White leg', treatment, 462
Wicket keeping, damage to fingers and thumbs during, 170, 171
Winter sports, injuries, 250–60
see also Ski-ing, snow
Women, athletes, and menstrual symptoms, 119
performance, 118
general medical care, 116–18
physique, improvement in, 113–15
representation at Olympic Games, 113
in sport, 113–22
see also Menstruation, Pregnancy
Wounds, gun-shot, accidental, 191–3

X-ray therapy, for bursal injuries, 331
deep, 309
Xylocaine, in injection therapy, 313

Zatopek, E., and training for peak, 85, 107

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